INDOOR AIR QUALITY: AROMATIC AND ALIPHATIC HYDROCARBONS AND THEIR HEALTH EFFECTS AT LOW LEVEL EXPOSURES

by

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ABSTRACT

Concentrations of volatile organic compounds (VOCs) were measured in eleven office buildings, previously characterised as sick buildings, in the city of Hobart, Tasmania. The principle aim of the study was to ascertain the types and concentrations of hydrocarbons found in non-industrial work environments, as well as determining the health effects caused by exposure to these low level compounds.

Two hundred and sixty five offices workers (136 males and 129 females) in selected public and private sector buildings were surveyed. The systematic building investigation consisted of measuring aromatic and aliphatic hydrocarbons, temperature, relative humidity and other building variables. In addition a self administered questionnaire assessed symptoms experienced while in the work environment, psychosocial factors and past disease history.

Hydrocarbon samples were collected on activated charcoal diffusion tubes and analysed using gas chromatography/mass spectrometry. Statistical methods, both parametric and non-parametric, highlighted correlations between variables and their levels of significance. These included the Chi-square test, correlation coefficients, and analysis of variance test.

Results indicated that individual hydrocarbon levels typically found in sampled buildings ranged from 0-1.758 mg/m^3 (1 week average). Weekly TVOC levels fluctuated and ranged from 0.012-1.934 mg/m^3. Types of VOCs identified included several alkanes, aromatics, halogenated hydrocarbons, alcohols and ketones.

Levels of VOCs varied seasonally. Higher concentrations were detectable in the winter months compared to summer. These values were affected by temperature, but not by humidity. Qualitative findings showed that symptoms typical of sick building syndrome were reported in 88.5% of respondents. Characteristic, and most frequently experienced symptoms included irritation of the eyes, nose and throat, cognitive effects, general manifestations, and cardiovascular effects. Psychosocial factors and other variables such as temperature and humidity had no significant effects on symptoms, but asthma and allergies did appear to influence symptom levels.

Individual species of VOCs influenced the types of symptoms reported, and VOC mixtures (TVOCs), were correlated with elevated retrospectively reported symptom levels ($r=.1789$, $<.05$). Sixty three percent of buildings were in the TVOC "no effects" range, while 36.4% were above 0.20 mg/m^3. Both SBS symptoms and sensory irritation were observed at levels below 0.20 mg/m^3.

Buildings were categorised as sick or healthy buildings on the basis of the VOCs in the air. It appears that specific organic compounds, especially those of the aromatic/aliphatic class, and the quantity detected are critical in determining a

building's potential to cause adverse health effects.

The TVOC concept was examined as a generic indicator to sensory irritation and sick building syndrome, as well as a review of current standards and guidelines in relation to low dose exposures to VOCs.

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LIST of ABBREVIATIONS

ABC -	Australian Building Code
ABCB -	Australian Building Code Board
ACGIH -	American Conference of Governmental and Industrial Hygienists
AGA -	Australian Gas Association
ANOVA -	Analysis of Variance
ANZECC -	Australian and New Zealand Environment and Conservation Council
AS -	Australian Standard
ASHRAE -	American Society of Heating, Refrigeration and Air Conditioning Engineers
BRE -	Building Research Establishment
BREEAM -	Building Research Establishment Environmental Assessment Method
BRI -	Building Related Illness
°C -	Degrees Celcius
CEC -	Commission of European Communities
CEM -	Centre of Environmental Medicine
COHb -	Carboxyhemoglobin
COMEAP -	Committee on the Medical Effects of Air Pollutants
CPSC -	Consumer Product Safety Commission
CPSU -	Community Public Sector Union
CSIRO -	Commonwealth Scientific Research Organisation
CSL -	Central Science Laboratory (University of Tasmania)
DEST -	Department of Environment, Sports and Territories
DH -	Department of Health
DNA -	Deoxyribonucleic Acid
DOE -	Department of Environment
DPIWE-	Department of Primary Industries, Water and Environment
ECA -	European Concerted Action Group
ECIETC -	European Chemical Industry Ecology and Toxicology Centre
EPAQS -	Expert Panel on Air Quality Standards
ETS -	Environmental Tobacco Smoke
FID -	Flame Ionisation Detection Method
GC/MS -	Gas Chromatography and Mass Spectrometry
HCHO -	Formaldehyde
HP -	Hypersensitvity Pneumonitis

HPLC -	High Performance Liquid Chromatography
hr -	Hour
HSE -	Health and Safety Executive
HVAC -	Heating, Ventilation and Air Conditioning System
IAP -	Indoor Air Pollution
IAQ -	Indoor Air Quality
IARC -	International Agency for Research on Cancer
IEH -	Institute for Environment and Health
IgE -	Immunoglobulin E (A Class of Antibody)
IR -	Photoacoustic Infrared Method
ISO -	International Standards Organisation
LC50 -	Lethal Concentration Limit Value
MAC	Maximum Allowable Concentrations
mbar	Millibars
MCS -	Multiple Chemical Sensitivity
mg/m^3 -	Milligram Per Metres Cubed
MRC -	Medical Research Council
MVOC -	Microbial Volatile Organic Compound
NATO/CCMS -	North Atlantic Treaty Organistaions Committee on the Challenge of Modern Society
NCBR -	Nordic Committee on Building Regulations
NHMRC -	National Health andMedical Research Council
NIOSH -	National Institute of Occupational Safety and Health
NKB -	Nordic Committee on Building Regulations
NOHSC -	National Occupational Health and Safety Commission
NOHSO -	National Occupational Health and Safety Office
OCE -	Office of the Commissioner for the Environment
OSHA -	Occupational Safety and Health Administration
PAH -	Polyaromatic Hydrocarbons
PCA -	Property Council of Australia
PCP -	Pentachlorophenol
PEL -	Permissible Exposure Limit
PM_{10} -	Particulate Mattter with an Aerodynamic Diameter of Less than 10μm
PM_{25} -	Particulate Mattter with an Aerodynamic Diameter of Less than 2.5μm
PMN -	Polymorphonuclear Neutrophils
POM -	Organic Compounds Associated with Particulate Matter
PPB -	Parts Per Billion
PPM -	Parts Per Million
PPT -	Parts Per Trillion

PPTV -	Parts Per Trillion Volume
PSU -	Public Sector Union
PTR-MS -	Proton Transfer Reaction Mass Spectrometry
SA -	Standards Australia
SBI -	Danish Building Research Institute
SBS -	Sick Building Syndrome
SDSC -	Standards Development Standing Committee
STEL -	Short Term Exposure Limits
SVOCs -	Semi Volatile Organic Compounds
TDR -	Tasmanian Development Resources
TSCA -	The Toxic Substances Control Act
TSP -	Total Suspended Particulate Matter
TVOC -	Total Volatile Organic Compound
TWA -	Time Weighted Averages
$\mu g/m^3$ -	Micrograms Per Metre Cubed
$\mu g/m^2$ -	Micrograms Per Metre Squared
UK -	United Kingdom
USA -	United States of America
USEPA -	United States Environmental Protection Agency
USSR -	Union of Soviet Socialist Republics
UV -	Ultraviolet
VICEPA -	Victorian Environmental Protection Authority
VOCs -	Volatile Organic Compounds
VVOCs -	Very Volatile Organic Compounds
WHO -	World Health Organisation
WHO/EURO -	Who Regional Office for Eastern/Western Europe and North America
WSA -	Workplace Standards Authority
yrs -	Years

CHAPTER ONE

INTRODUCTION

1.1 Indoor Air and Organic Gases

Until recently, volatile organic compounds (VOCs) in the indoor environment have received very little attention. New technologies available to analyse atmospheric components have made it possible to measure these pollutants accurately, yet most available studies focus on the outdoor environment rather than the internal atmosphere in buildings. Investigating the range of VOCs in indoor air is arduous given their large number, variable physico-chemical properties and multiple sources.

The potential health effects associated with long and short term exposure to chemicals belonging to the VOC class are of most concern. This is especially the case for compounds identified as possessing carcinogenic, neurotoxic, mutagenic and toxicological properties. Determining a correlation between VOCs and potential health effects has proven difficult. Effects from exposures are usually subtle with nonspecific transient symptoms that are not identifiable as a clearly defined disease. To add to the complexity, no proven biological marker exists in individuals making diagnosis difficult.

In most cases ambient levels of volatile organic compounds are several orders of magnitude below current Australian and overseas standards. Even though these standards are not a measure of relative toxicity, they insinuate that low concentrations should elicit only a minor effect on human health. Field studies in large population groups have shown the contrary, where concentrations well below recommended standards exhibit odourant properties, and toxic and irritative effects. The widespread use of such chemicals suggests that these chronic low level exposures need to be characterised to better understand their impact on human health and wellbeing.

1.2 Thesis Background

Aromatic and aliphatic hydrocarbons are suggested to be carcinogens (Maroni *et al.* 1995, Sigma-Aldrich 1996, Snyder 1987). Unfortunately the actual health consequences of most organics found in the indoor air are not at present well defined.

Only recently have indoor air quality investigations recognised the significance of VOCs, and as a consequence studies have been scarce. The ubiquitous nature of VOCs means that they can be found in all building components including construction, manufacturing of materials, furniture and maintenance. It is the deterioration of these chemically unstable products, or

pollutant sources, that subsequently release contaminants into the indoor environment by a form of evaporative process.

Sources can be frequent with a constant emission, as is the case with gas and cigarettes, an episodic source with declining emission (i.e. latex paint), or a constant source with declining emission, like chipboard based furniture. This "outgassing" of pollutants can continue rapidly or slowly over many years depending on the properties of the chemical in question. The primary emissions and chemical transformations that result from these processes suggest that the health effects of exposures need to be determined.

Few Australian studies have been conducted characterising VOCs and the general principles governing exposure. The exception to this is published work relating to formaldehyde (Brown 1996, Dingle & Smith 1994, Garret *et al.* 1997a, Garret *et al.* 1997b) and VOCs in homes (Brown *et al.* 1994). Examination of the office environment has been even less extensively investigated (Dingle & Olden 1992, Rowe & Wilke 1994, Williams 1992). In Australia, health based goals for VOC exposure in non-industrial environments have been derived by reviewing information from outside Australia, and from unpublished and published VOC measurements (NHMRC 1989).

As one might expect, more extensive information comes from other countries like the United States, Italy, and the Nordic countries who dominate work done on VOCs (Andersson *et al.* 1997, Cooper *et al.* 1995, Crump *et al.* 1997, Molhave *et al.* 1997, Otto *et al.* 1992, Raaschounielsen *et al.* 1997, Santos *et al.* 1997). Overall, these VOC investigations comprise a significant range of study topics including material emission tests, indoor air quality modelling, and exposure studies. Research has already been undertaken on known amounts of VOCs in carefully controlled conditions, leading to statistically significant results between symptoms experienced and VOC exposure (Koren & Devlin 1992, Koren *et al.* 1992, Molhave *et al.* 1986, Otto *et al.* 1992). These studies have subsequently led to the extension and development of validated analytical methods for the quantitative characterisation of effects of solvents on humans.

Tasmania, Australia's smallest state, in particular, has been greatly neglected from an indoor air quality perspective. Results of a Hobart study undertaken in 1995 (Mesaros 1995) showed that the office environment is having a significant impact on the health of employees. Ninety percent of sixty one buildings showed signs of sick building syndrome (SBS), with employees frequently experiencing a high level of sensory irritation associated with the indoor environment.

1.3 The Purpose of the Thesis

Results of the Tasmanian study (1995) warranted further investigation into the possible causes of sensory irritation in employees while in these buildings. Symptoms highlighted in the qualitative results were indicative of VOC exposure.

Therefore, the principle aim of this thesis is to ascertain the concentration and types of volatile organic compounds found in non-industrial work environments, and to determine if these low background level VOCs are having a measurable effect on the health of employees. The hypotheses underlying this research program are:

> the health effects caused by chronic low level volatile organic compounds have been underestimated. This is reflected by the lack of toxicological analyses and regulation on acceptable levels to low exposures. These organic gases (especially aromatic and aliphatic hydrocarbons) are conducive to increased sensory irritation in individuals while in the indoor environment, and are a key pollutant in buildings identified as suffering from "sick building syndrome".

To test this hypothesis, the following objectives have been set:

1. To assess the types and concentrations of hydrocarbons found in the indoor office environment;
2. To evaluate if the types and concentrations of hydrocarbons vary on a weekly or seasonal basis;
3. To determine and verify chronic low level human exposure to aromatic and aliphatic hydrocarbons, and the subsequent health effects, while in the indoor environment;
4. To test a relationship between volatile organic compound levels and work related morbidity;
5. To examine both the "Total Volatile Organic Compound" concept and low individual concentrations of hydrocarbons as generic indicators of sensory irritation;
6. Review current standards and guidelines in relation to low dose exposures to volatile organic compounds in non-industrial environments (offices); and
7. To outline and evaluate approaches used in reducing volatile organic compound concentrations in the indoor environment.

The investigation was undertaken in 11 office buildings already classified as suffering from SBS. A control building was incorporated into the study (with no

evidence of SBS) to provide a comparison. Even though the number of buildings sampled are not numerous, they do represent a good cross section of office accommodation in Tasmania. From an epidemiological viewpoint, the study can be termed as extensive, given the size of population sampled. Therefore outcomes are applicable to indoor populations elsewhere.

1.4 Definition of Terms

The term "Volatile Organic Compound (VOC)" has been used extensively throughout this thesis. Although widely used, the abbreviated term VOC has been incorrectly applied in some technical literature to represent all organic compounds irrespective of their classification (defined by boiling point range). The World Health Organisation's (WHO) definition of VOC has been chosen for this thesis, and is categorised by organic compounds with a boiling point ranging from 50 °C to 260 °C (WHO 1989).

When VOCs are referred to within the thesis, it incorporates all volatile compounds in indoor air (unless otherwise specified) and includes aliphatic hydrocarbons which may be straight, branched chain, or cyclic, aromatic hydrocarbons, halogenated hydrocarbons (primarily chlorine or fluorine), and oxygenated hydrocarbons such as aldehydes, alcohols, ketones, esters, ethers, and acids.

The term "outgassing" is also used. This refers to the slow release of chemicals within the structure of the material. These might be components of the material itself, or various additives, such as solvents, softeners, dyes and treatments, or they may be by products of reactions going on within, or at the surface of the material e.g. oxidation.

When discussing health effects, the nomenclature used essentially follows conventional toxicological terminology. Generally, "exposure" refers to the contact with external environment media containing the chemical or chemicals of interest, unless stated otherwise. "Deposition" refers to the capture of the chemical at the body surface site. This can be either on the skin, respiratory system or gastrointestinal tract. The term "dose" is used to mean the amount of chemical deposited on or translocated to a site on or with the body where toxic effects take place.

Other terms are defined within the thesis and in the list of abbreviations.

1.5 Overview of Thesis Structure

The thesis is organised to follow a sequential framework beginning with general indoor air quality issues followed by a discussion on specific aspects relating to volatile organic compounds. A review of related literature is provided throughout the thesis. Chapters Two to Six essentially serve as a background for general issues significant to indoor air quality assessment. Chapters Seven to Eleven contain the experimental work and discussion of results. The thesis is then concluded with Chapter Twelve suggesting recommendations and control of the indoor environment.

When discussing VOCs it is nearly impossible to isolate them from other indoor air quality factors because of their interactive qualities. Chapter Two therefore outlines the nature, sources and toxicity of physical, biological and chemical pollutants found in indoor air. Air in the home and work environment is of equal, if not of greater significance to human well-being than outdoor air pollution. Certainly it is known that personal exposure to many common air pollutants is driven by indoor exposures. Energy conserving designs used after the 1970s caused basic design features to be modified at the detriment to health. Modern building technologies unknowingly introduced potentially harmful pollutants which are greatly varied in their sources. These include a wide variety of building materials, consumer products, building maintenance materials and office equipment.

Only pollutants that have an association with VOCs are reviewed in this thesis. This is done to illustrate particular points, and to demonstrate their potential to cause adverse health effects in humans. Although many more indoor pollutants exist their relationship with VOCs has not yet been demonstrated. Variables such as temperature and humidity are included in this chapter even though they are not conventionally defined as a physical, biological or chemical pollutant. These two factors have been shown to influence the intensity and distribution of VOCs, therefore their inclusion as an extraneous factor seems appropriate.

Chapter Three provides an overview of the health effects from pollutants including conventional methods of exposure assessment and their inherent weaknesses. A major objective of indoor air investigations centre around implications to health, therefore exposure pathways and resulting consequences to physical wellbeing are evaluated, with a focus on the difficulties of finding relationships between pollutants measured and resulting symptoms. This is an issue of concern predominant in VOC studies. Correlations between low level carcinogenic substances and resultant symptoms among individuals have proved

difficult to quantify. In addition, it is still not clear if individual or mixtures of contaminants provide the greater health risk. At present, the major tenet of systematic building studies indicate that sensory irritation reported by individuals may be due to combined effects of many VOCs found in indoor air, rather than exposure to a single compound.

Aside from quantitative analysis of a substance, the probability of disease or death from any exposure (biological, chemical or physical) is a integral part of indoor studies. The four distinct areas of risk assessment (hazard identification, dose-exposure assessment, exposure assessment and risk characterisation) will be discussed and evaluated for their suitability in VOC analysis (Maroni *et al.* 1995).

Organic compounds' relative abundance, gas-phase presence, and potential to cause sensory irritation are important considerations when scrutinising indoor air chemistry. Chapter Four details types of organic pollutants and their physico-chemical properties, with a special reference to aromatic and aliphatic compounds. Early studies in indoor air (De Bortoli *et al.* 1986, Johansson 1978) identified hundreds of organic compounds, yet insufficient data prevented conclusions on population exposures. Subsequent knowledge on the occurrence of organic compounds increased, with more field surveys being undertaken to determine frequency distributions of concentrations. At present available data on semivolatile organic compounds (SVOC) and organic compounds associated with particulate matter (POM) have received the most attention due to their widespread diffusion and sanitary significance. VOCs on the other hand have had limited quantification, with even fewer data relating to non-industrial environments.

The issue of standards of organic pollutants are subsequently analysed in Chapter Five. To date legislation, standards and guidelines in relation to VOCs have been limited. Exposure standards for atmospheric contaminants in the occupational environment have little relevance to non-industrial workplaces. Short term exposure limits (STEL) and time weighted averages (TWA) for airborne contaminants are inadequate guidelines when examining chronic low level organic compounds. Alternative scientific views on acceptable exposure ranges to VOCs have not reached a unanimous finding, with the exception of the TVOC concept. The TVOC theory provides a dose-response relationship between VOC exposure and air acceptability providing a tentative framework for human exposure studies.

Chapter Six provides a review of VOC investigations conducted, which is integral to the background of this study. It includes the development of VOC studies and provides an overview of the three conventionally used methodologies (systematic building investigations, exposure studies and emission tests) applied in

VOC research. It also highlights the qualitative and quantitative techniques applied to various VOC investigations.

Chapter Seven describes the methodology used in this thesis. To fully evaluate VOC concentrations and their health effects a combination of state of the art qualitative and quantitative testing procedures were used. VOC concentrations were measured using personal sampling devices and symptomatology assessed by questionnaire method. Variables such as temperature and humidity were also evaluated as they are thought to influence the behaviour of VOCs.

The study took place over ten weeks, five weeks in summer and five weeks in winter, to explore possible weekly and seasonal differences in VOC concentrations. Quantitative samples were analysed using Gas Chromatography (GC)/Mass Spectrometry (MS). There is a general trend toward using this technique over flame ionisation detection (FID) methods, because of its increased accuracy. Compounds were analysed on the basis of their prevalence in indoor air, toxicity and carcinogenicity, therefore only those of the VOC class (particularly aromatics, aliphatics and halogenated hydrocarbons) were investigated. The advantage in selecting compounds from within this category is that direct comparisons could be made to the few existing VOC studies. In order to demonstrate significant relationships between hydrocarbon concentrations, SBS symptoms, and dose-response effects, a range of parametric and nonparametric statistical techniques was used.

The experimental results are examined in Chapter Eight, Chapter Nine and Chapter Ten, and introduce new information on volatile organic compounds and their health effects. Chapter Eight focuses on individual volatile organic compounds and their mixtures, whereas Chapter Nine focuses on general SBS symptoms and sensory irritation. Chapter Ten then proposes a relationship between the two. Chapter Eleven provides a discussion of the results, debating the feasibility of TVOC as an effective risk index, the toxicological effects of aromatic/aliphatic hydrocarbons and a review of environmental standards.

Chapter Twelve discusses options for the control of VOCs in the indoor climate, especially in the elements of design, construction and maintenance. Future studies and a standardised way of assessing risks of VOCs are suggested to clarify the relationship between exposure to volatile organic chemicals and illhealth in individuals. The final part of this chapter concludes the thesis by reviewing the hypothesis and the findings.

This thesis contributes much needed information on the background levels of VOCs present in the non-industrial indoor environment. It is not only

complementary to existing overseas studies on organic compounds but provides a new Australian perspective on indoor environments.

CHAPTER TWO

NATURE, SOURCES and TOXICITY of POLLUTANTS

2.1 Introduction

The internal atmosphere in buildings is a complex and dynamic system containing a large variety of interacting chemical compounds in both the gas and particulate phases. The pollutants present in the indoor environment include physical, biological and chemical contaminants. Interactions between pollutant sources, sinks, air movement between rooms and buildings, the outdoors and individual activity patterns determine individual exposures. This is particularly significant when evaluating primary emissions, binary mixtures and complex synergistic effects (three or more constituents) between VOCs and various other indoor pollutants.

It has been well established that VOCs are released into indoor air by almost all materials, consumer products, furnishings, and fuels. Identified sources of VOCs, and the more important classes of compounds emitted by these sources, are detailed in Chapter 4 and Chapter 6. The main focus of this chapter is to illustrate how the dispersion, concentration, toxicity and sources of VOCs can be altered in the presence of other contaminants. Therefore the outcomes of these potential reactions should be taken into consideration when monitoring and evaluating VOCs in indoor air investigations, such as the Tasmanian study discussed in this thesis.

For example, reactions among indoor pollutants can produce products that otherwise might not be present in the indoor environment. According to Weschler and Shields (1997), chemical reactions between ozone, nitric oxide, nitrogen dioxide and selected unsaturated hydrocarbons are capable of generating various free radical reactions, essentially creating additional chemical compounds. In some instances the source of VOCs is not clear, and VOC concentrations measured could be the result of byproducts produced from other pollutant interactions, such as VOCs of a microbial origin. To add to the complexity of quantifying VOCs, many of the VOC emissions (primary and mixtures) are difficult to detect using sampling and analytical techniques currently applied to indoor air.

The relationship between VOCs and other indoor air pollutants makes characterisation of the indoor air difficult to define, but modern measuring techniques of outdoor ambient air pollution have provided a means of assessing a range of sources and levels of exposure.

2.2 Interactions Between VOCs and Other Indoor Pollutants

2.2.1 PARTICULATE MATTER

Recent studies have renewed interest in the health effects of submicron particles. The most extensive data on sources and health effects have been derived from studies on outdoor air limited to the measurement of total suspended particulate matter (TSP), respirable particles PM_{10} (particulate matter with a aerodynamic diameter less than 10 μm) and inhalable particles PM_{25} (<2.5 μm).

Combustion sources are the most recognised generator of fine-mode particles, and these contain a range of organic and inorganic material. Other sources of finer particles include biological contaminants and cooking aerosols, whereas coarse-mode fractions are largely carried from outdoors e.g. dust and larger biological fragments. In commercial buildings, such as offices, where there is no smoking or cooking, the major sources of submicron particles is outdoor air.

These small solids have enormous surface areas on which chemical reactions can be catalysed by a variety of effects (Demirabo *et al.* 1997, Raush *et al.* 1995). Sulphur dioxide is one such example which has a more toxic effect in the presence of particles like coal dust and tobacco smoke (Maroni *et al.* 1995). This is especially the case for volatile organic compounds, where there is evidence of synergism between particulate matter and polyaromatic/aliphatic hydrocarbons (Gil & Adonis 1996). Polycyclic aromatic hydrocarbons (PAH) are of particular concern because of their carcinogenic potential (Maroni *et al.* 1995) and, according to WHO (1987), once PAHs enter the air, they can be absorbed on particles and inhaled into the lungs. It has also been stated that in certain particles, some biologically active compounds may be absorbed inside of particles (Chao *et al.* 1998), resulting in the particles themselves becoming toxic.

The toxic effects of particulates depends on how deeply they penetrate the respiratory system, what fraction is retained, and how well the body can cope with the toxic agents. Particulates in the range of <2.5 μm are considered the most dangerous, because they will be retained deep in the lung (primarily the tracheo-bronchial and pulmonary region), while the larger particles are principally trapped in the nasopharyngeal region (Amdur *et al.* 1991). The same particles (<2.5 μm) are thought to play a major role in the soiling processes and also contribute to corrosion, current leakage and shorts in electronic equipment (Weschler *et al.* 1996).

Overall, the limited studies on particulate matter have been unable to fully examine the prevalence and possible range of health effects associated with submicron particles and VOCs.

2.2.2 DUST

A significant source of indoor particulates is wind-borne dust. Dust may contain natural inorganic and biological particles, including pollen and road dust. Depending on particle size, dust may settle locally (as seen in plate 1) or may spread throughout entire buildings.

PLATE 1

Residual dust from an air conditioning vent
(Location: Glenorchy police station 1995)

It has been postulated that chemical reactions occur between dust and gases. The gases are subsequently absorbed onto particles, producing new species of compounds. These hybrid mixes (in particular VOCs × particulates) are believed to contribute to SBS symptoms and accelerated mortality (Bates 1995). According to Wolkoff *et al.* (1997), it is difficult to quantify but new analytical methods are being developed to measure these oxidative and reactive species.

Dust may also directly transmit VOCs from one area to another distributing contaminants which may as a result either increase or decrease human exposure.

Airborne dust particles have the potential to transfer and contain polycyclic aromatic hydrocarbons. An example of this is highlighted by Lewtas *et al.* (1997), who state that dust particles, especially those <2.5 μm, contain PAHs which alter DNA adduct levels in white blood cells at low to moderate exposures.

2.2.3 ENVIRONMENTAL TOBACCO SMOKE (ETS)

Another pollutant with strong VOC associations is tobacco smoke. As with all forms of smoke, it consists of a mixture of solids, liquids, and gases. The composition of tobacco smoke (table 2.1) is fairly well known as well as its health effects.

According to Meyer (1993), the chemical composition of the smoke include 27 different alkanes, 51 alkenes and alkynes, 96 aromatic hydrocarbons, 15 sterols, 24 alcohols, 272 esters, 45 aldehydes or ketones, 1 quinone, 30 nitriles, 57 acids, sulphur compounds, 55 phenols, 107 alkaloids, 14 amino acids, 17 inorganic salts and other ashes.

Tobacco emissions, and more recently incense condensates (Chen & Lee 1996), are an important air contaminant and source of volatile organic compounds. According to Hoffmann *et al.* (1997), the presence of smokers causes respirable suspended particulates (RSP) to be three to twelve times higher indoors than outdoors, exposing others to involuntary smoking especially aromatic hydrocarbons. Although smoking is prohibited in most office buildings (but rarely in factories or workshops), cigarette smoke may still enter buildings by outdoor smoking facilities being inappropriately located at or near airconditioning intakes. Therefore, any quantification of VOCs and related contaminants must consider potential influences from ETS.

2.2.4 MOULDS AND FUNGI

Moulds and fungal spores are associated with air borne particles and indoor air. The indoor environment can potentially place human occupants at greater risk than the outside environment because inadequate air filtration and excessive indoor humidity (Godish *et al.* 1996) have the potential to create contamination with these bioaerosols (Robertson 1997). Plate two, illustrates this point by highlighting the extent to which some offices investigated in this Tasmanian study, are affected by mould and fungal deposits.

TABLE 2.1

Distribution of constituents in fresh, undiluted mainstream smoke and diluted sidestream smoke from nonfilter cigarettes

(Source: Maroni *et al.* 1995, p129)

Constituent	Amount in MS	Range SS/MS
Vapour Phase		
Carbon monoxide	10-23 mg	2.5-4.7
Carbon dioxide	20-40 mg	8-11
Carbonyl sulfide	12-42 µg	0.03-0.13
Benzene	12-48 µg	5-10
Toluene	100-200 µg	5.6-8.3
Formaldehyde	70-100 µg	0.1-.50
Acrolein	60-100 µg	8-15
Acetone	100-250 µg	2-5
Pyridine	16-40 µg	6.5-20
3-Methylpyridine	12-36 µg	3-13
3-Vinylpyridine	11-30 µg	20-40
Hydrogen cyanide	400-500 µg	0.1-0.25
Hydrazine	32 ng	3
Ammonia	50-130 µg	3.7-5.1
Methylamine	11.5-28.7 µg	4.2-6.4
Dimethylamine	7.8-10 µg	3.7-5.1
Nitrogen oxides	100-600 µg	4-10
N-Nitrodimethylamine	10-40 ng	20-100
N-Nitrodiethylamine	ND-25 ng	<40
N-Nitrosopyrrolidine	6-30 ng	6-30
Formic acid	210-490 µg	1.4-1.6
Acetic acid	330-810 µg	1.9-3.6
Mehtyl chloride	150-600 µg	1.7-3.3
1,3-Butadiene	69.2 µg	3-6
Particle phase		
Particulate matter	15-40	1.3-1.9
Nicotine	1.2.5	2.6-3.3
Anatabine	2-20	-0.5
Phenol	60-140	1.6-3.0
Catechol	100-360	0.6-0.9
Hydroquinone	110-300	0.7-0.9
Aniline	360	30
2-Toluidine	160	19
2-Naphtylamine	1.7	30
4-Aminobiphenyl	4.6	31
Benz(a)anthracene	20-70	2-4
Benzo(a)pyrene	20-40	2.5-3.5
Cholestrol	22	0.9
y-butyrolactone	10-22	3.6-5.0
Quinoline	0.5-2	3-11
Harman	1.7-3.1	0.7-1.7
N-Nitrosononicotine	200-3000	0.5-3
N-Nitrosodiethanolamine	20-70	1.2
Cadmium	110	7.2
Nikel	20-80	13-30
Zinc	60	6.7
Polonium	0.04-0.1	1.0-4.0
Benzoic acid	14-28	0.67-0.95
Lactic acid	63-174	0.5-0.7
Glycolic acid	37-126	0.6-0.95
Succinic acid	110-140	0.43-0.62
PCDDs and PCDFs	1	2

MS - Mainstream smoke SS - Sidestream smoke

PLATE 2

Acoustic tiles affected by water damage and fungal growth
(Location: Glenorchy police station 1995)

Components of ventilation systems are the primary sources of pathogenic microorganisms which subsequently transmit spore byproducts to building occupants. Volatile organic compound emissions are thought to be the result of these secondary metabolites (MVOCs). Several volatile compounds have been found in building air, and in the majority of cases were traced back to colonised fibreglass duct liners in the HVAC system (Ahearn *et al.* 1996). MVOCs are believed to be responsible for elevated aldehyde and ketone levels found in buildings. Building investigations examining organic compounds should therefore include biologic toxins as a possible source of VOC contamination.

While the ecology of moulds is not fully understood, growth and emissions of MVOCs on air filters are suspected to contribute to health complaints (Schleibinger *et al.* 1997, Wessen & Schoeps 1996), with secondary metabolites giving rise to a variety of symptoms typical of SBS. Volatile metabolites, rather than exposure to the organism itself, can cause acute toxic diseases as well as long term genotoxic and carcinogenic effects at low concentrations (Samet 1988). An alternative view is

presented in some studies where high microbe concentrations had no apparent adverse effect (Jaffal *et al.* 1997).

2.2.5 INORGANIC POLLUTANTS

Inorganic pollutants (carbon dioxide, carbon monoxide, nitrogen dioxide, sulphur dioxide and ozone) generated from outdoors occur in indoor air in small amounts. These pollutants have the potential to change rapidly and reach significant levels depending on how contaminants infiltrate the indoor environment. Some sources of inorganic pollutants include complete and incomplete combustion products from a variety of origins including domestic energy use, as a byproduct of ETS, and human exhalation. At present, the only confirmed inorganic pollutant that has the potential to interact with volatile organic compounds is ozone.

Ozone is a pulmonary irritant that effects the mucous membranes and other lung tissues, causing fibrosis and respiratory function failure. Examples of indoor sources of ambient ozone include copying machines, UV lighting, laser printers and electrostatic cleaners. According to Maroni *et al.* (1995), the presence of hydroxyl radicals and volatile organic compounds in the atmosphere (of natural or anthropogenic origin) causes a shift in the atmospheric equilibrium generating higher levels of ozone. Reiss *et al.* (1995) extend on this point by stating that complex photochemical reactions occur between polar organic compounds, nitrogen oxides and ozone. These elevated ozone levels produce increases in unsaturated hydrocarbon concentrations and visa versa. As with many other indoor pollutants the reactions between chemicals subsequently create byproducts of unknown morphology and toxicity (Weschler & Shields 1997).

2.2.6 ORGANIC POLLUTANTS

A great variety of organic materials have been identified in indoor air. These include aliphatic and aromatic hydrocarbons, pesticides, polynuclear aromatic hydrocarbons, polychlorinated biphenyls, formaldehyde and various ketones and aldehydes.

According to the World Health Organisation (WHO 1989), organic pollutants are classified in four major categories, these nomenclatures are:

1. The very volatile organic compounds (VVOC) - Boiling point range <0°C - 50°C;
2. The volatile organic compounds (VOC) - Boiling point range 50°C - 100°C to 240°C - 260 °C;
3. The semivolatile organic compounds (SVOC) - 240°C - 260°C to 380°C - 400°C; and

4. The organic compounds associated with particulate matter and organic matter (POM) > 380°C.

Although there are no defined limits between these arbitrary categories (WHO 1989), they are useful in determining the absorbent required for collection of compounds. As a result concentration and exposure data are available by category (Maroni *et al.* 1995). Information on SVOCs and POM are scarce, whereas data on PAHs, pesticides and polychlorinated biphenyls are more extensive because of their relevance to population health. Chemicals of the VOC class, on the other hand, have received little attention (until recently) and consequently limited concentration and exposure data exists.

Examination of the VOC class of organic pollutants has been selected for this thesis because the VOC category encompasses substances with a wide variety of physico-chemical properties which are relatively abundant in indoor air. These substances can be emitted from various sources found in the office environment including construction materials, wood products, carpets, textiles, personal products, cleaning products, paints, petroleum based coating materials (Guo *et al.* 1998), and gas appliances.

The practicality of measuring VOCs was another consideration for this study. Unlike many other organic chemicals (semivolatile organic compounds), the required instrumentation and analytical methods needed for VOC sampling were easily available. In addition, VOCs readily absorb on Tenax, carbon molecular black or charcoal, and therefore are suitable for personal VOC exposure sampling in a large scale epidemiological survey.

The ability of organic chemicals to cause health effects varies greatly from those that are highly toxic, to those with no known health effect. As with other pollutants, the extent and nature of the health effect will depend on many factors including level of exposure and length of time exposed. Eye and respiratory tract infection, headaches, dizziness, visual disorders, and memory impairment are among the immediate symptoms. At present not much is known about the health effects caused by the entire range of organic compounds, but many are proven to cause cancer in animals, some are suspected of causing, or are known to cause cancer in humans e.g. benzene and tetrachloroethylene.

Interactions between organic pollutants and their derivatives have aroused some interest over the years. Chemical reactions amongst organics is possible, even without catalysts, in indoor air. It is hypothesised that multiple sources of VOCs, in particular, exemplify a second class of mixtures believed to be even more toxic than primary emissions, but the complex mixtures produced are at present unknown.

2.3 Extraneous Factors

2.3.1 TEMPERATURE AND HUMIDITY

Even though extraneous factors such as temperature and humidity are not categorised as an indoor pollutant, their influence on VOCs is indisputable.

Overall, the rate of chemical reactions have been found to be faster in VOCs with increased temperatures. According to Norback (1995), associations have been found between varying room temperature and VOC concentrations present. Vanderwal *et al.* (1997) extend on this point by examining the influence of temperature on material emission rates. They found that the effect of temperature on VOC emission rates influence chemical reactions. In all cases the total emitted mass of VOCs increased with temperature, and bake-out periods of only a few days may be unsuccessful in sufficiently outgassing materials.

It is well documented that humidified air in buildings increases perceived bad air quality (Reinikainen *et al.* 1997). It has also been established that humidity is similar in effect to temperature, with higher VOC levels being recorded in areas of higher humidity (Smedje *et al.* 1997). Specifically, humidification affects the decomposition of nonchlorinated hydrocarbons and VOC structures (Futamura *et al.* 1997).

Humidity can influence quantitative analysis of VOCs as well (Vanderwal *et al.* 1998). Goss and Eisenreich (1997) elaborate on this point by stating that the sorption of VOCs onto traps are greatly decreased with increased relative humidity, making VOC analysis particularly susceptible to climatic parameters.

2.4 Conclusion

In addition to the indoor contaminants mentioned, VOCs may be influenced by the presence of additional indoor pollutants. The limited studies undertaken on indoor environments in general have not characterised these relationships yet, therefore information is not readily interpretable. That is not to say they do not exist. Systematic building studies and exposure studies have successfully attempted to evaluate the inherent association between VOCs and other building variables. Perhaps the advent of new and improved techniques for quantifying VOCs will reveal previously unknown additive and multiplicative effects.

CHAPTER THREE

HEALTH EFFECTS of INDOOR POLLUTION

3.1 Introduction

There is no question that the physical and chemical conditions of air inside buildings can interfere with normal body functions. Contaminants can reach sensitive tissues in the human body causing discomfort, loss of function, and changes in structure leading to disease.

This is especially the case with volatile organic compound exposure. It has been shown that medium to low level VOCs have the potential to cause a nonhomogeneous conglomeration of specific and nonspecific complaints among building populations, ranging from mild irritation to debilitating health effects.

VOCs are also believed to be the main aetiology behind sick building syndrome (SBS). Reasons for this include the fact that many VOCs have the potential to cause both sensory irritation and nervous system symptoms characteristic of SBS. They are more often than not found in higher concentrations indoors compared to outdoor concentrations, and because of the quantities present in indoor air, may cause symptoms as a result of additive and combined effects.

The Tasmanian study (systematic building investigation) discussed in this thesis focuses largely on the health effects caused by VOC and TVOC exposure. Therefore it is an integral part of VOC research to be familiar with the toxicity and metabolism of organic chemicals so associations can be made between measured VOC/TVOC levels and simultaneously reported symptoms. To examine this further, the following chapter provides a review of the current published literature drawing on the fields of toxicology, occupational health and medicine.

As the data-base for VOCs in indoor air is still in its infancy, and to date has no formal risk management strategy of its own, it is critical to understand the fundamental philosophy and principles adopted by current occupational health and safety strategies. Therefore this chapter outlines the processes involved in identifying health effects, the development of exposure standards, and how VOCs fit into existing controls of health hazards, especially when characterising risk.

3.2 Exposure Pathways

Exposure represents contact between a concentration of a chemical agent, or other materials, and the person, or population of interest. Familiarity with the patterns or pathways for exposure is critical in VOC analysis, particularly in assessing its toxicity. By knowing the mechanisms by which humans perceive and respond to chemical contaminants, it is possible to evaluate and predict responses to specific compounds. Many factors influence exposure, but none more important

than dose received by the individual. The magnitude of the dose depends on a number of factors, these are:

1. The volumes ingested or inhaled;
2. The fractions of the inhaled material transferred across epithelial membranes of the skin, respiratory, and gastrointestinal tract;
3. The fractions transported via circulating fluids to target tissues; and
4. The fractional uptake by the target tissues.

Each of these factors have intersubject variability such as age, sex, and health status, as well as inherent variabilities as race and size.

In some cases the dose does not have to be great for absorption to occur, and appear very much to be dependent on a compound's chemical and physical characteristics. For example, Piotnowski (1971) found that human volunteers inhaled and subsequently absorbed 60-80% of phenol (a common commercial disinfectant and solvent) after exposure to a low level dose. In this case the phenol was subsequently biotransformed into the liver, which is the target organ for this compound.

With chronic or repetitive exposures, other factors affect the dose of interest. When a chemical's retention, or its effect on the target tissues, is cumulative and clearance or recovery is slow, the dose of interest can be represented by cumulative uptake. This storage of toxins occurs when the rate of exposure is greater than the rate of metabolism or elimination, causing a accumulation of chemicals. Benzene exposure is one example. According to Sarto *et al.* (1984), increases in structural chromosome aberrations were observed in workers exposed to average benzene concentrations ranging from 1.9-5.3 ppm. No previous history of high level benzene exposure was noted in subjects, therefore chromosomal alterations were caused by a cumulative effect to chronic low levels. Exposures such as this have the potential to cause more long term health effects which can be cross generational. This is increased when exposure is to a genotoxic and mutagenic substance. Shy (1993) states that this is especially important when exposures contain mixtures of VOCs, which even at low doses have the potential to disturb reproductive functioning and cause cancer.

3.2.1 THE RESPIRATORY SYSTEM

The surface and systemic uptake of chemicals from the inhaled air depends both on the physical and chemical properties of the chemicals and the person's pattern of respiration. Compounds in the form of gases and vapors, like VOCs encountered in indoor air, rapidly contact airway surfaces by molecular diffusion. Surface uptake is limited for compounds that are relatively insoluble in water e.g.

ozone. For chemicals such as ozone, the greatest uptake can be in the lung periphery (as seen in figure 3.1) where surface areas are the greatest. For more water soluble gases, dissolution or reaction with surface fluids on the airways facilitates removal from the airstream. Vapours that are highly soluble, such as sulphur dioxide, are almost completely removed in the airways of the head and very little vapour penetrates into the lung airways (Ellenhorn & Barceloux 1990).

The depth of penetration increases with decreasing particle size. Thus, larger particles are retained throughout the bronchial tree and the greater the diameter, the greater the chance that they will not reach the alveoli. Smaller particles are retained in decreasing amounts but practically all small particle deposition takes place in the alveoli (figure 3.1), hence why VOC gases have been implicated in reduced respiratory functioning.

As the respiratory system is the organ directly affected by air pollution, investigation on the health effects of exposure have been diverse. A consensus of findings reveal that effects associated with exposure to low level VOCs and general chemical pollutants cause or exacerbate conditions predominantly in the lower airways, such as coughing, wheezing, shortness of breath, and phlegm.

For example, in chamber exposure studies lasting between 1-6hr, Sundell and Zuber (1996) found that chemical reactions of VOCs and nitrogen oxides caused respiratory symptoms such as lung function decrements, increased airway responsiveness and inflammatory reactions. These effects occurred even at the lowest concentration tested at 160 $\mu g/m^3$ for 6hr.

3.2.2 THE GASTROINTESTINAL SYSTEM

Ingestion may also contribute to the uptake of chemicals that were initially inhaled since material deposited on, or dissolved in, the bronchial mucous blanket is eventually swallowed (Ballantyne *et al.* 1995). Although there is no specific literature regarding the ingestion of VOCs from the indoor environment, it would be unreasonable to eliminate it as a potential exposure pathway. Many volatile organic compounds have been identified as mucous membrane irritants, and inhalation followed by ingestion onto the mucosal linings of the stomach is inevitable.

According to Ballantyne *et al.* (1995), unless the ingested material itself affects the tract, any systemic response depends upon the absorption through the mucosal cells lining the lumen. Although absorption may occur anywhere along the length of the tract the main region for effective translocation is the small intestine (figure 3.2).

The enormous absorptive capacity of the intestinal mucosa result in a large total surface area for absorption. Although passive diffusion is the main absorptive process, active systems also allow for the uptake of toxic chemicals.

FIGURE 3.1

Illustration of the respiratory system
(Source: Hurst 1984, p115)

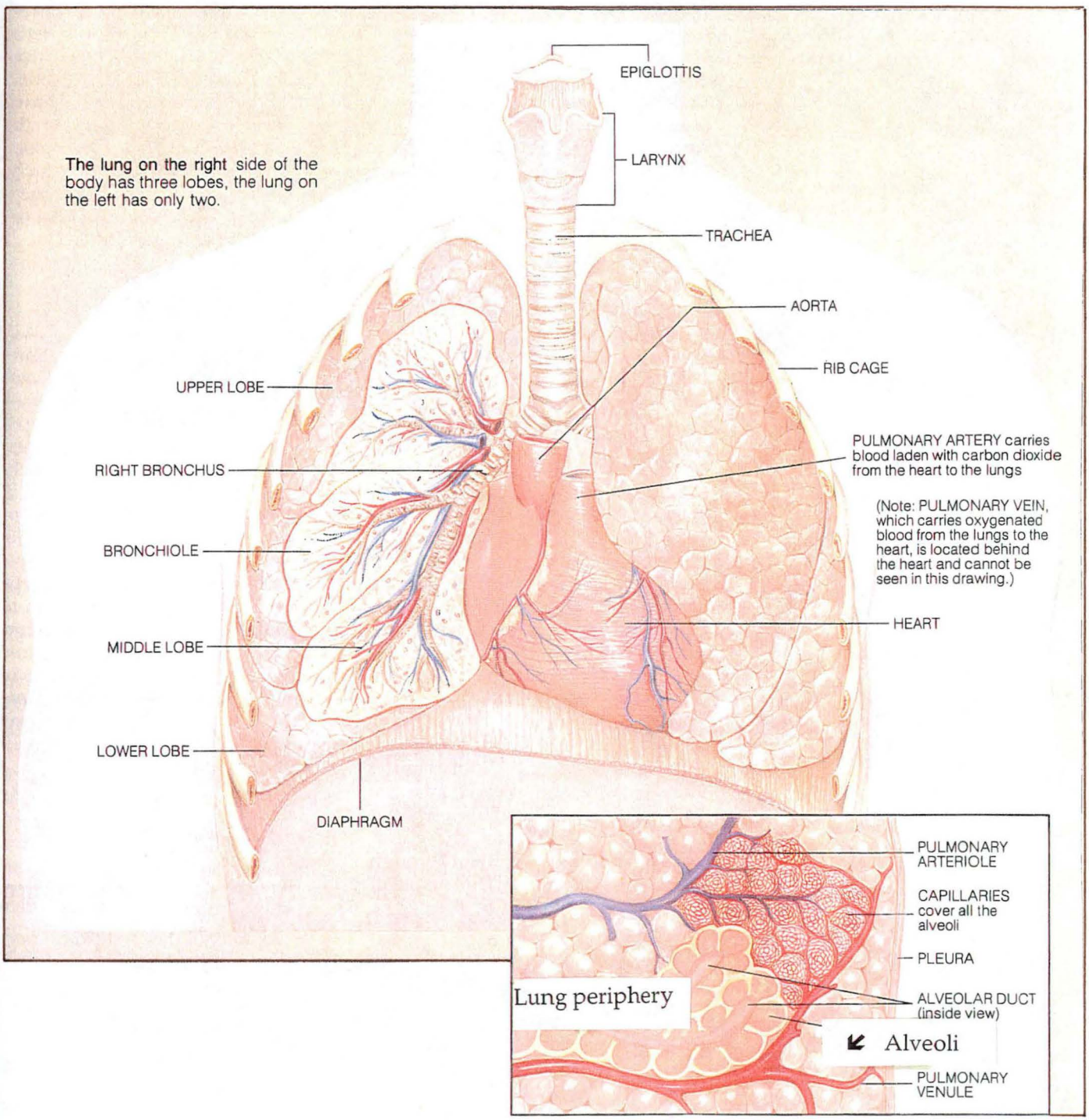
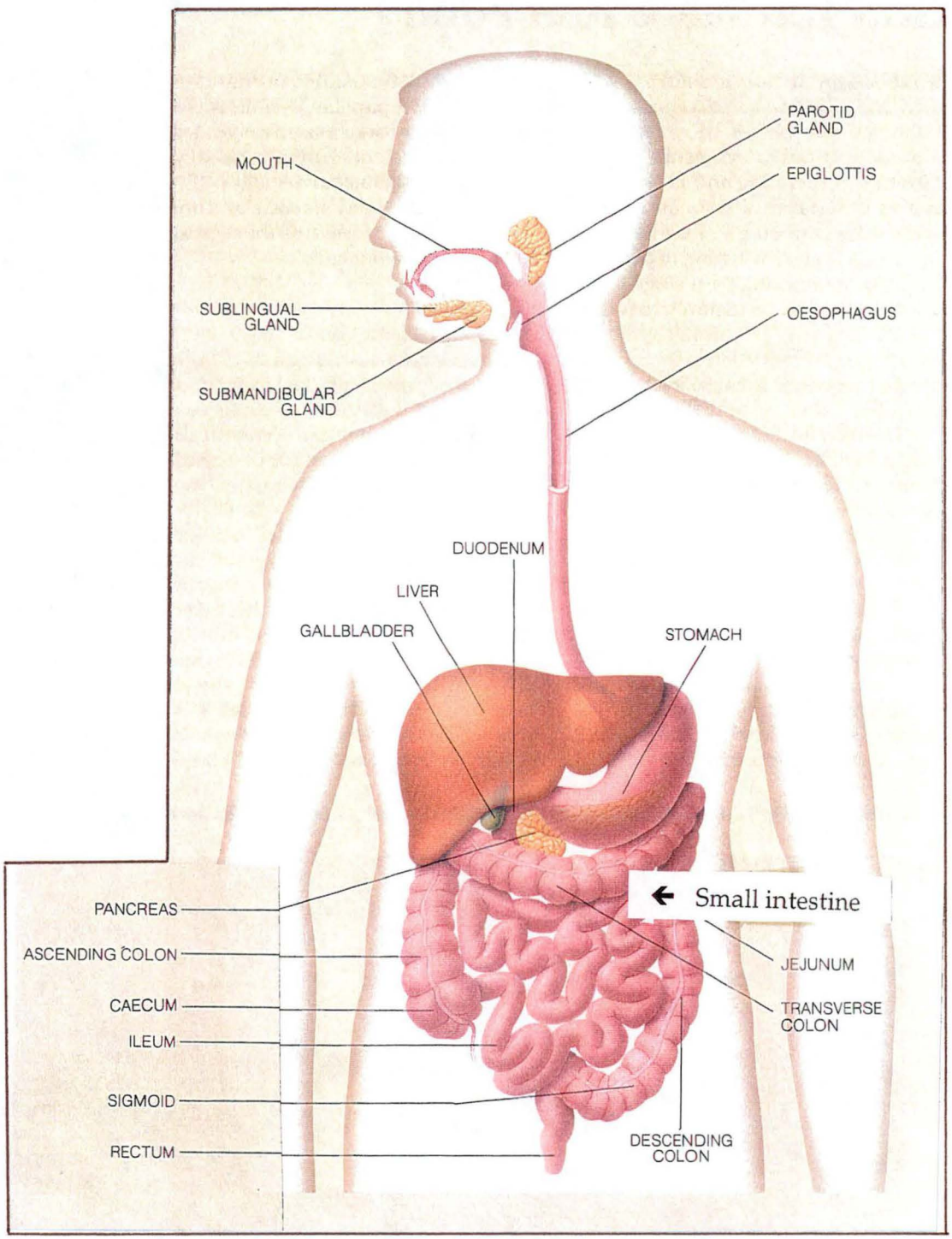


FIGURE 3.2

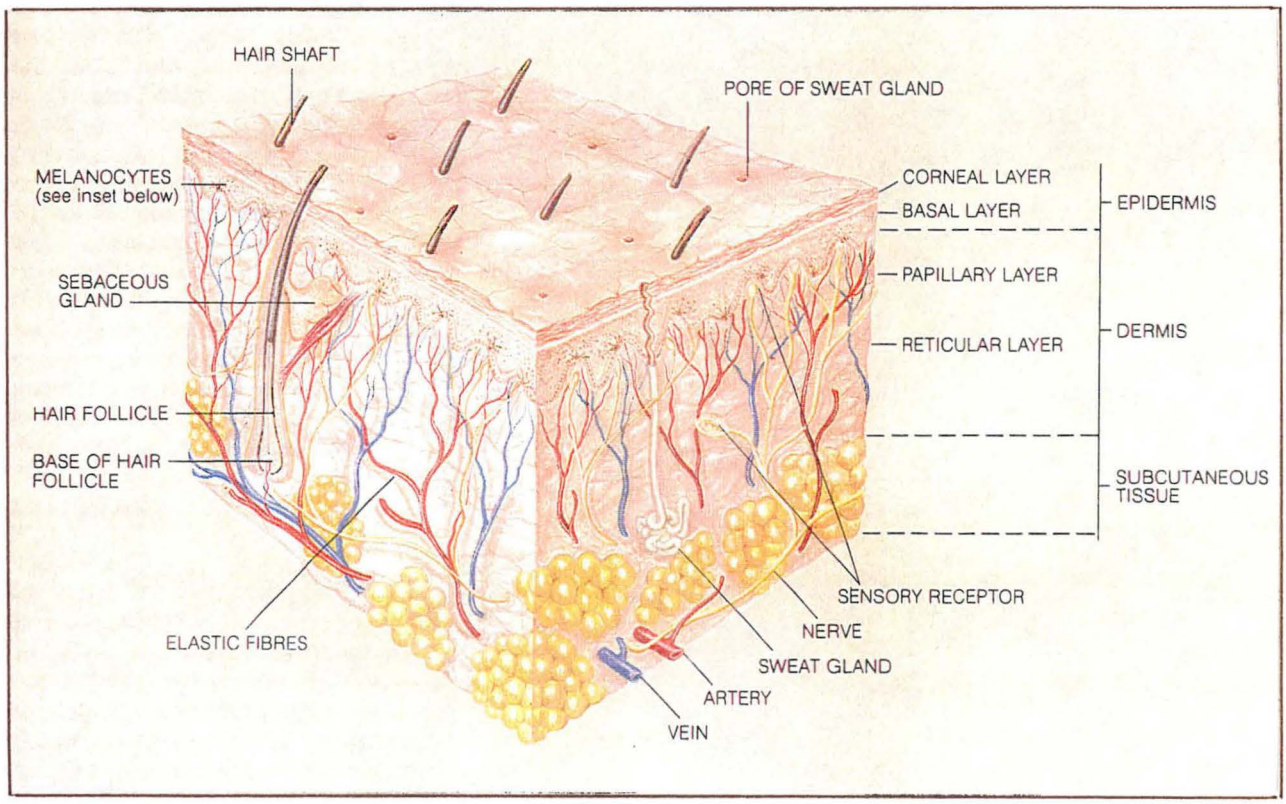
Diagrammatic representation of the gastrointestinal system
(Source: Hurst 1984, p235)



In addition, materials absorbed from the gastrointestinal tract can enter either the lymphatic system or the portal blood circulation. The latter carries matter to the liver from which it may be actively excreted into the bile. This is especially relevant to VOCs which have known effects on the blood and liver, such as benzene, 1,2-dichloroethane, trichloroethylene, acetone and carbon tetrachloride.

FIGURE 3.3

Structural layers of the skin.
(Source: Hurst 1984, p133)



3.2.3 DERMAL EFFECTS

The skin is generally an environmental barrier against entry of environmental chemicals, and is one of the foremost routes of exposure. In order to be absorbed via this route (percutaneous absorption) an agent must traverse a number of cellular layers before gaining access to the general circulation (Amdur *et al.* 1991). As illustrated in figure 3.3, the epidermis represents the primary barrier to percutaneous absorption. The dermis located just under the epidermis, is subsequently permeable to many materials diffused by the top layer of skin.

The main factors that affect percutaneous absorption are the degree of lipid solubility of the chemicals, site on the body, local blood flow, and skin temperature. The exposure to substances on the skin surface can also penetrate additional pathways such as hair follicles, sweat glands, and sebaceous glands increasing overall dermal uptake (Amdur *et al.* 1991). Some chemicals are readily absorbed through the skin such as phenols and carbon tetrachloride, while others alter the integrity of the skin and facilitate penetration of other materials by increasing permeability. According to Brooks and Riviere (1996), the absorption and penetration of phenol into tissues through the skin are greater with the presence of certain compounds like ethanol. These combined effects are also seen in coal tar derivatives which can reach the systemic circulation through skin absorption. The resulting chemical can react with selected wavelengths of natural or artificial light causing dermatitis and other health effects.

Generally exposure to many of VOCs found indoors like phenol, 1,1,2,2-tetrachloroethane, and limonene, would certainly be absorbed percutaneously. But Sato and Nakajima (1978) claim that rarely would any of these compounds be absorbed in sufficient quantities during normal exposure to produce toxicity.

3.3 Specific Health Effects

3.3.1 EFFECTS ON THE RESPIRATORY SYSTEM

Many effects on the respiratory system have been associated with indoor pollutant exposure. According to Maroni *et al.* (1995), these include acute and chronic changes in pulmonary function, increased incidence and prevalence of respiratory symptoms, acute worsening of pre-existing respiratory symptoms, and sensitisation of the airways. Symptoms characteristic of respiratory susceptibility include coughs, wheezing, upper respiratory tract infections, colds, influenza, and pneumonia. Of all the indoor pollutants, PM_{10} particulates and their VOC counterparts (such as those measured in this Tasmanian study), are especially believed to cause increases in respiratory symptoms (Janssen *et al.* 1998).

In extreme cases some gases are simple asphyxiants when present in high concentrations. Carbon dioxide is one such example, but is also known to cause toxic effects at concentrations which do not cause asphyxiation (Maroni *et al.* 1995).

3.3.2 SENSORY EFFECTS

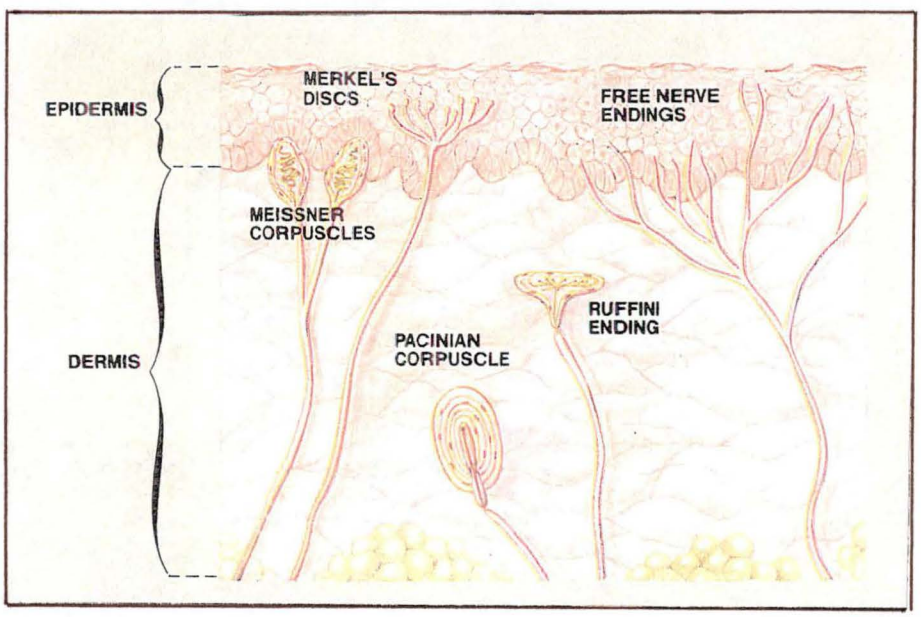
Sensory effects are very common on exposure to indoor pollutants, and are propriated through the neural system, resulting in sensory dysfunctions. These

effects can affect the body through direct and indirect mechanisms. Direct exposure to a compound stimulates the senses outside the body, such as experiencing odour, and inside the body by evoking irritation to the organs (Maroni *et al.* 1995). Even though the two sensory responses act quite differently, they are difficult to separate because exposure to a contaminant has the potential to stimulate both systems simultaneously (Amdur *et al.* 1991).

It is believed that the mechanisms primarily responsible for provoking irritation are the free endings of the trigeminal nerves (Godish 1995) located on the facial skin, nasal cavity and other skin areas (figure 3.4).

FIGURE 3.4

Free nerve endings of the skin
(Source: Hurst 1984, p138)



These nerves can be aroused by both chemical and physical agents. According to Aviado and Salem (1995), specific responses to chemical inhalants immediate a response known as the Kratschmer reflex consisting of apnea, bradycardia and a rise and fall in aortic blood pressure. General stimulation can result in irritation described as burning, stinging, or smarting. It also has the capability of inducing changes in the heart rate, coughing, sneezing, and respiratory frequency. Shy (1993) has even demonstrated neurobehavioural effects which were displayed from sensory excitation to low environmental exposures.

Exposure studies into TVOCs have determined that trigeminal systems play a significant role in reactions to organic pollutants. A study by Comettomuniz *et al.* (1997) highlighted that as the number of chemical mixtures increase so do the

effects from their physicochemical properties. Therefore VOCs, and their aggregates, can be seen as significant in the determination of sensitivity, and particularly in evoking irritation. This is elaborated on in a study by Hau and Connell (1998), which suggests that acetates, alcohols, ketones probably bind to a common receptor site located in the hydrophobic interior of the lipid bi-layer membrane of the olfactory cilia, and that odour thresholds of related VOCs appear to share a common receptor site which may be additive, causing sensory effects.

But other factors can stimulate the sensory systems as well as chemical contaminants. For example, Fang *et al.* (1998) found that sensory effects and perception of indoor air quality were significantly influenced by extraneous variables like temperature and humidity.

3.3.3 IRRITATIVE EFFECTS

Irritative effects are characteristic of indoor air complaints, especially in sick building syndrome. Irritation can target any part of the body causing a wide variety of symptoms including eye, nose, and throat irritation, sneezing, coughing, general discomfort, inflammation, soreness, itchiness, grittiness, redness, headaches, nausea and other physiological responses (WHO 1989).

VOCs are proven to be irritating in themselves, and are believed to heighten the effects of mucosal irritants. In a study by Comettomuniz and Cain (1998) it has been found that human nasal and eye irritation increased when exposed to minute amounts of M-aliphatic alcohols and 1-octanol, and slight general irritation upon exposure to minute VOC mixes (Rothweiler & Schlatter 1993). Hundell *et al.* (1992) therefore believes that it is quite feasible that subthreshold levels of VOCs may interact additively or hyperadditively stimulating trigeminal nerve receptors. Wolkoff *et al.* (1998) uses a more specific example and states that both the volatile and non-volatile fraction of cleaning agents are suspected to irritate the airways.

The potency and mixture of VOCs also influence the levels of irritation experienced. In a study undertaken by Molhave *et al.* (1986), elevations in eye and throat irritation were evident when test subjects were exposed to 22 VOCs (25 mg/m^3). VOC mixtures therefore appear to have a strong relationship between exposure concentration and irritative effects. Alternatively, Fanger (1998) found temperature and humidity to be a significant causative agent in irritative symptoms.

3.3.4 CARDIOVASCULAR EFFECTS

Only rarely has contact with indoor air highlighted cardiovascular effects. According to Maroni *et al.* (1995), only carbon monoxide and ETS has been implicated in cardiovascular symptoms and in changes in cardiovascular disease,

morbidity and mortality. ETS via an active smoker, is documented to cause chronic pulmonary diseases and cardiovascular disease (Witschi *et al.* 1997), increased heart rate, increased blood pressure, cancers, tachycardia and increased blood carboxyhemoglobin (COHb). Both ETS and carbon monoxide compounds have an affinity for combining with hemoglobin, resulting in a reduction of the oxygen carrying capacity of tissues with the highest oxygen demand e.g. heart and brain. Therefore a raise in COHb levels is considered to be a potential health hazard.

3.3.5 ALLERGENIC RESPONSES

Notable triggers for allergic diseases are allergens derived from house dust mites, animal dander, moulds and protein containing furnishings e.g. feathers. Only recently has the tremendous increase in the genesis of allergies and asthma been equated with elevated concentrations of VOCs (Andersson *et al.* 1997). Kreiger *et al.* (1998) believe that VOCs are capable of provoking asthmatic crises either by a direct pollutant effect or by potentialising the allergenic response of the bronchi e.g. formaldehyde exposure. This has been supported by studies undertaken by Wieslander *et al.* (1997) and Kreiger *et al.* (1998), who found that formaldehyde and VOC emissions from newly painted surfaces caused inflammatory reactions in the lung and an onset of asthma.

Typical allergic responses range from rhinitis and nasal congestion to conjunctival inflammation, uticaria and asthma. Specific allergenic diseases such as extrinsic allergic alveolitis (hypersensitivity pneumonitis) are also caused by exposure to indoor pollutants (Maroni *et al.* 1995). Rebouts of this interstitial lung disease are exacerbated by airborne allergens and contaminated air conditioning and humidification systems.

General pollutants found in indoor air have the additional capability of changing the body's immune system by lowering its defences against inhaled particles. People may become sensitised to a particular allergen, causing extreme immunological dysfunction when exposed to even small amounts of the offending allergen (Roitt *et al.* 1989). Such chemicals, like formaldehyde, can cause this specific immune response in some individuals. Maroni *et al.* (1995) state that these substances "sensitise" people, and exposure to a sensitiser, once sensitisation has occurred, may mainfest itself as a skin rash or asthmatic condition. In some individuals this reaction can be extremely severe. In addition it is believed by authors such as Dietert and Hedge (1996), that these immune changes are related to inflammatory based reactions from low environmental exposures.

An alternative view is held by Gustafsson *et al.* (1996), who claims that no definite conclusions can be made about allergy and hypersensitivity reactions because of the interactions with other factors. Chang *et al.* (1993) are of the same

opinion, but state that the contaminant itself may not cause the reaction but merely exacerbates a pre-existing condition.

3.3.6 CARCINOGENIC EFFECTS

Monitoring of inorganic and organic compounds has brought to attention the toxicological effects of chemicals on cells and tissues.

The majority of chemicals found indoors are thought to be carcinogenic or mutagenic. It has therefore been argued that the accelerated increase in cancer has been attributed, in part, to VOC exposure. According to Badman and Jaffe (1996), metabolites of benzene and other volatile hydrocarbons are implicated in the causation of leukemia's and other cancers of the blood. Tap *et al.* (1996) extend on this by claiming that particular chemicals, like toluene, have also shown varying degrees of cell degeneration in tissue samples. For example, chlorinated hydrocarbons have been shown to possess genotoxic and cytotoxic potentials (Tafazoli *et al.* 1998). Shy (1993) confirms this view and states that organic compounds have been proven to have extensive neurotoxic, reproductive, and carcinogenic effects. ETS and wood smoke are also suspected to be responsible for increased mutagenicity in humans by causing adenocarcinoma (Hoffmann *et al.* 1997).

Of all the organic pollutants polycyclic aromatic hydrocarbons are believed to be the worst offenders toxicologically (Perera 1997). According to Bishop *et al.* (1997), five to ten percent of birth defects are due to exposure to a teratogenic agent, with PAHs posing the greatest risk. Parental occupational exposure is important when evaluating carcinogenic effects. Cordier *et al.* (1997) elaborates on this point by stating that childhood brain tumors are an example where the history of solvent exposure in parents is strongly correlated with subsequent cancer development in offspring.

Overall, because of the long latency problem between complex mixtures and carcinogenic effects, further research results are unlikely to yield data of adequate quality so broader conclusions can be made about the wider population (Shy 1993).

3.4 Syndromes Related to Indoor Air Quality

3.4.1 SICK BUILDING SYNDROME

The term "sick building syndrome" (SBS) describes a range of symptoms reported by building occupants, that are associated with their presence in a building. It is characterised by occupants of the building having an extra number of irritative symptoms from the eye, nose and throat and lower airways, skin

reactions, unspecific hypersensitivity reaction, mental fatigue, headache, dizziness, whilst they are in the premises (Muzi *et al.* 1998, Ooi *et al.* 1998, WHO 1989). To add to the complexity of SBS its recognition as an actual diagnosable disorder has not been confirmed, unlike building related illness.

Sick building syndrome is usually recognised when the causes of the complaints are "unknown", coupled with the fact that building occupants feel better soon after leaving the building. Kroling (1998) states that evidence of SBS is only detectable in systematic building investigations, which are then compared to controls. Therefore, this Tasmanian study fulfils the criteria required for investigating sick building syndrome. Firstly, occupant symptomatology and VOC concentrations are measured in a range of office buildings (thought to be "sick") which are then compared to a control (no reported health complaints), and secondly, it is a systematic building investigation because it attempts to determine (quantitatively and qualitatively) whether VOC/TVOC levels (and other building factors as well e.g. air exchange rates) are associated SBS symptoms in buildings.

Generally when a substantial proportion of those spending time in a building report or experience acute on site discomfort, sick building syndrome should be suspected. An alternative view to this argument is held by many authors, such as Bullinger *et al.* (1998), Lahtinen (1998), Ooi and Goh (1997), and Seeber *et al.* (1998), who claim that SBS is a stress related disorder which should be dealt with from an occupational stress perspective. Contrastingly, researchers like Rothman and Weintraub (1995) claim that SBS is predominantly influenced by temperature and humidity variables, and not by stress or chemical pollutants alone.

3.4.2 BUILDING RELATED ILLNESS

Building related illness is identified as a set of symptoms that can be attributed directly to specific airborne building contaminants. Legionnaires' disease and hypersensitivity pneumonitis are classified as building related illnesses, and sources of exposure are predominately of biological origin.

Legionnaires' disease is one of the most noted of all indoor illnesses. *Legionella pneumophila* has been found in many building components particularly cooling towers, humidifiers and tap water (Maroni *et al.* 1995). Symptoms are similar to those with sick building syndrome exposure, but differ in severity. It is essentially a virulent form of pneumonia characterised by headache, chest pain, lung congestion and high fever.

Hypersensitivity pneumonitis (HP), including humidifier fever, is also a lung disease similar to pneumonia (Burge 1982). According to Fink (1998), sources of infection have been traced back to air conditioning systems and humidifiers contaminated with moulds and bacteria. Symptoms emerge shortly after exposure and include coughs, dyspnea, chills, myalgia, fatigue and high fever. Because

moulds are one of the principle agents, there is no evidence to suggest that MVOCs are not involved in inducing HP.

Humidifier fever's exact origin is unknown, but its similarity to hypersensitivity pneumonitis makes it worthy of mention. It shares the same symptoms as HP, with additional effects like high attack rate, flu like symptoms, malaise and short term effects. It has been closely related to bacteria and fungi colonies in air conditioning systems, and is thought to cause a variety of allergenic responses.

3.4.3 MULTIPLE CHEMICAL SENSITIVITY

Multiple chemical sensitivity (idiopathic environmental intolerance) is becoming more widely equated with indoor air pollutant exposure and VOCs (Miller 1997, Ross 1997, Winterbauer 1997). Individuals with this syndrome are said to suffer from multi-system illness as a result of contact with a range of contaminants, even at low levels. Dyer (1997) states that the worst offending substances thought to cause onset of illness include natural gas, pesticides, solvents, new carpet, renovation materials, glues, copy paper, cleaning products, combustion products, petrochemicals, and any organic solvent.

According to Zeim and McTamney (1997), disorders commonly seen in chemically sensitive individuals are headache, chronic fatigue, attention deficit, chronic respiratory inflammation, and musculoskeletal aching. More long term injury that magnifies the response to exposures can include neurogenic inflammation, time dependent sensitisation, and immune activation (Sorg & Prasad 1997). Weiss (1994) extends on this by claiming that chemical sensitivity in the population has more to do with SBS than originally thought. This view has been supported in a study undertaken by Lax and Henneberger (1995), who found that the onset of multiple chemical sensitvity was related to solvent exposures in indoor air (Bell *et al.* 1997). Overall knowledge about the precise causes and mechanisms through which this disease is manifested is limited.

3.4.4 SOLVENT ENCEPHALOPATHY

Solvent encephalopathy describes a cluster of symptoms such as headache, irritability, difficulty concentrating, and fine motor deficits caused by exposure to solvents including those of the VOC class. According to Maroni *et al.* (1995), solvent encephalopathy occurs upon exposure to VOCs which are at levels below threshold limit values for industrial solvents. Overall, there is a relationship between duration of exposure and the time required to stop symptoms after exposure ceases. In a study undertaken by Varney *et al.* (1998), intermediate levels of organic solvents have been shown to cause injury to the subcortical white matter

in neuropathological studies. Alternatively, Morrow *et al.* (1996) demonstrated cognitive and mood changes following solvent exposure in test subjects. There is no evidence at present to suggest that the levels of VOCs present in office and home environments are not having a similar effect.

3.5 Risk Assessment

A formal methodology to quantify risk, especially to low level pollutants like those found in indoor air, provides a valuable tool to make decisions about regulating internal environments. The four components involved in risk assessment are as follows:

1. Hazard identification - this determines whether exposure to a compound can cause adverse health effects;
2. Dose-response effects - this equates a relationship between the dose of a substance and its subsequent health effect;
3. Exposure assessment - which predicts what exposures are likely to affect the population at large; and
4. Risk characterisation - which estimates the risks involved in exposure and suggest ways of reducing those risks.

3.5.1 HAZARD IDENTIFICATION

Identification of hazards is critical if appropriate standards are to be introduced to protect people from the adverse health effects from exposure. This is more so for substances identified as suspected or established carcinogens, such as the VOCs found in indoor air. It is also useful in determining which toxins are worth controlling, rather than simply focusing on those singled out for attention by particular laws or public controversies.

The exposure standards for the occupational environment, currently practiced in Australia (NOHSC 1995) highlight the hazardous potential of exposure to a range of atmospheric contaminants, believed to present a health hazard. The exposure standards only consider absorption via inhalation. Both carcinogenic and non-carcinogenic substances, like airborne particulates including organic and inorganic chemicals are listed, and their evaluation as a hazard has been established because there is sufficient information to suggest that an exposure standard is appropriate. For example, the hazard identification for carcinogenic substances in Australia have been based on the findings by the International Agency for Research on Cancer and Carcinogenisis (ECIETC 1986). According to

the NOHSC (1995), not all substances have been included, but this does not imply that they have non-hazardous status, but insufficient information exists to assign an exposure standard.

The National Occupational Health and Safety Commission (NOHSC) have adopted three categories originally established by The Commission of the European Communities (CEC) to identify hazardous materials. This system of classification is used to indicate the strength of the causal association between these substances and the development of cancer (NOHSC 1995). These are:

1. Category One (Established Human Carcinogens) - are those substances known to be carcinogenic to humans. There is sufficient evidence to establish a causal association between human exposure to these substances and the development of cancer;
2. Category Two (Probable Human Carcinogens) - are substances for which there is sufficient evidence to provide a strong presumption that human exposure might result in the development of cancer. This evidence is generally based on appropriate long term animal studies, limited epidemiological evidence or other relevant information; and
3. Category Three (Substances Suspected of Having Carcinogenic Potential) - are those substances which have possible carcinogenic effects on humans, but in respect of which the available information is not adequate for making satisfactory assessment. There is some evidence from appropriate animal or epidemiological studies, but this is insufficient to place the substance in category two.

There are many organic compounds found in indoor air that are proven or potential carcinogens. Compounds such as benzene, toluene, styrene, and carbon tetrachloride are all confirmed human carcinogens. Exposure standards for atmospheric contaminants in the occupational environment are useful in assisting control of occupational air pollution, but limitations in this form of exposure guidance is that low dosage or no effect levels cannot be confidently identified. In addition, chemical agents that are toxic or highly toxic are not included. An example would be agents like *n*-decane, *n*-octane, α-pinene and ethanol which are commonly found indoors. Therefore full interpretation of hazard identification for building variables, like low level VOCs is still incomplete.

3.5.2 DOSE RESPONSE RELATIONSHIPS

Dose-response relationships provide the second tier in the risk assessment procedure, and is usually seen as vital in assessing pollutants. In practice a work environment may contain a number of airborne contaminants and exposure to these

additional substances, either individually, simulatneously or sequentially could give rise to an increased hazard to health.

"Dose assessment" of a received compound may require personal monitoring be complemented by biological monitoring to take full account of such factors as absorption by routes other than inhalation, biological variations and personal habits. For example, a topical study undertaken by Brooks and Riviere (1996) examined quantitative percutaneous absorption on mice to see if the dose of binary mixtures of phenolic compounds at various concentrations gave rise to systemic toxicity. In another study by Eide and Zahlsen (1996), various doses of aromatic hydrocarbons (75, 150, 300 and 450ppm), were given to mice, via inhalation, to determine its toxic effects. Results produced from such animal tests are then extrapolated to humans in constructing estimates of risk resulting from environmental exposures, but uncertainties in results are evident in this form of cross species extrapolation.

The evaluation of the "response" factor is much more difficult. Chronic toxic effects to substances usually manifest themselves sometime during the period of exposure making dose-response relationships more defined. This is especially the case for compounds in which the health effects arise only hours after a exposure to a substance. Maroni *et al.* (1995) uses humidifier fever to illustrate this point. The symptoms of humidifier fever typically occur 4-8hr after exposure on the first day back at work after the weekend, but resolve within 24hr despite continuous exposure the disease does not occur until after the next weekend. Carcinogenic compounds on the other hand may take a substantial amount of time from the initiating event to a clinical expression of disease, extending the emergence of a response. A diagnosis of cancer may not be made until long after cessation of exposure which may take a few years. This is demonstrated by the IARC (1989), who claim that an acute peak in formaldehyde exposure is more important in the development of nasal tumors than accumulated doses, even though the development of the tumor may take years.

As demonstrated in many other fields of toxicology, exposure to smaller or low level compounds are not often evaluated in great detail. In addition, examination of low concentrations of single pure, additive, synergistic and potentiated (when the chemical has an effect but the second chemical does not, but enhances the effect of the former chemical on a combined exposure) substances are neglected. Principle agents found in indoor air fit this category, combined with the fact that doses are usually both chronic and acute. It is therefore implicit that efforts are made to demonstrate the toxicity, carcinogenicity and health effects of VOC agents, otherwise substances with the potential to harm man will continue to be missed (Doll 1985).

3.5.3 EXPOSURE ASSESSMENT

Exposure studies are incredibly complex because of the number of different factors all interrelating and operating at the same time. Usually the components of exposure assessment include the population likely to be exposed, sources of contamination, exposure pathways, concentrations or mixtures of agents, and susceptible populations.

Exposure studies use a variety of techniques, and are one of the principle means of assessing exposure to indoor air contaminants. In particular, many exposure studies have been used to evaluate the health effects from VOC and TVOC exposures. Authors such as Molhave *et al.* (1986), Molhave (1990), Molhave *et al.* (1993), Hundell *et al.* (1992), Otto *et al.* (1990), and Otto *et al.* (1992), have used exposure studies extensively to measure general and irritation effects from exposure to defined concentrations of organic compounds. Exposure studies are also used in a number of inhalation experiments to determine allergenic effects (Koren *et al.* 1992).

These assessments are then used to establish the distribution of exposures and resulting doses in a given population, based on known contact with or in proximity to materials believed to have caused effects. An example of this is the TVOC dose-response relationship model (Molhave 1990), where exposure studies determined the effects of VOCs. In this model, certain concentrations of VOCs are believed to give rise to a series of symptoms, and air acceptability, in the general population. From these investigations inferences are made about the range of integrated exposures experienced by various population groups given specific assumptions from dose response relationships.

The setting of standards for the concentrations found in ambient air are then based on the human exposure surveys.

3.5.4 RELATIVE RISK

Estimates of risk is the final component of the risk assessment procedure. Predicting and reducing risk is based on the evaluations made in the other three categories already outlined. To discover the "risks", conclusions are made about the cause of a hazard by monitoring its effect to estimate the level of exposure that produces the highest additional risk of disease that is socially acceptable. With this information, exposure limits are introduced in the form of standards or guidance notes to reduce the risk of exposure. Remedial action follows to improve or prevent the environment from precipitating further risk. In indoor environments the employer, building owner and manager, state and local government agencies are usually responsible for the implementation of this (Maroni *et al.* 1995).

Unfortunately, much of the risk assessment procedure is confounded by political and economic pressures.

At present, the only characterisation of risk is for chemical agents that do not specifically pertain to the indoor environment (Spengler 1993). The concern over carcinogens and mutagens, and the potential health risks posed by their presence has emphasised the adoption of indicators or levels below which risk is deemed as minimal. For example, exposure standards for airborne contaminants are expressed as time weighted average (TWA) over an entire eight hour working day. TWA exposures permit excursions above the exposure standard provided they are compensated for by equivalent excursions below the standard during the working day (NOHSC 1995). Short term exposure limits (STEL) provide guidelines for the control of short term exposure. STELs are established to minimise the risk of intolerable irritation, chronic or reversible tissue change and narcosis (NOHSC 1995). A category labelled peak limitation is included where STEL and TWA is seen as inappropriate. Peak limitation represents a maximum peak concentration to which individuals may be exposed (NOHSC 1995).

These conventional indicators of risk, as with most theoretical frameworks, neglect to include long term, non-industrial and low dose relationships such as those found in indoor air. Many of the chemical pollutants are known to seriously affect health, but the long term risks associated with exposure has been underestimated. This is illustrated in a series of studies undertaken by Wallace (1986) and Tancrede *et al.* (1987), who examined the cancer risk for benzene, carbon tetrachloride, chloroform, styrene, 1,1,1-trichloroethane and α-pinene, based on measurements, in New Jersey and California, and a study of 300 Dutch homes. This is illustrated in table 3.1, which summarises the mean individual risk estimated from total VOC indoor exposure for each of these studies. Depending on whether established or extrapolated potency factors were used, the risk varies by a factor of ten overall.

TABLE 3.1

Individual life time cancer risk estimates per 1000 for
indoor air pollutants (VOCs)
(Source: Spengler 1993, p20)

VOC Study	High end	Expected
New Jersey (n=9 VOCs)	19-30	3
Californian (n=19 VOCs)	2	1
Dutch (n=44 VOCs)	1-2	.2-1

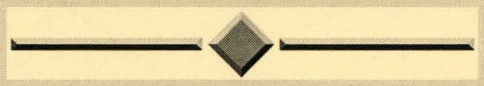
Charactersiation of risk, especially to indoor pollutants, needs further development in its methods and should emphasise the diseases which are characterised by multifactorial aetiologies, multistage development with long latencies, and lack of clear linkage to causative agents. Then effective, preventative measures of control can be implemented.

3.6 Conclusion

Much of our understanding of the adverse effects of indoor pollutants come from the traditional paradigms of toxicology. Advances in the basic biological sciences, provided the underpinning for understanding the mechanisms by which physical and chemical compounds produce their adverse effects. The recognition of such information is an absolute requirement for evaluating appropriate regulations of pollutants, but its relevance to non-industrial indoor air is equivocal. The challenge still lies in assessing and measuring the diverse and variable characteristics of low dose exposures, including all components (both physicochemical and physiological) of an epidemiological investigation. This is particularly pertinent to the special elements found in VOCs, where the parameters describing the probability of illhealth are indistinct.

The apparent environmental risk to agents in the indoor air warrants further research efforts to be embarked upon to understand the basic underlying principles of environmentally related diseases.

CHAPTER FOUR



THE CHEMISTRY of VOLATILE ORGANIC COMPOUNDS

4.1 Introduction

Gradually, over the last decade, research emphasis has been placed on the spectrum of organic compounds found indoors. The large, ever-growing number of chemicals used in building construction and materials may be several times higher in concentration indoors than levels found outdoors (Albaigés *et al.* 1993). A broad range of organic compounds occupies the ambient air but none so interesting as the volatile organic class. VOCs' ubiquitous presence and health perspective makes them the most controversial pollutants indoors.

Data is incomplete on many VOC species, primarily because of the large number of compounds found, the low concentrations present and the limitations in detection methods. Therefore, knowledge and understanding of these compounds originate from the fundamental principles of organic chemistry.

To understand typical concentrations of VOCs it is essential to review studies done in the past. It is necessary to refer to the published findings of other surveys in order be familiar with what kind of compounds are expected to be found in indoor air, and at approximately what levels. This background information can then be used as a reference for other VOC investigations, such as this Tasmanian study, especially when it comes to selecting the most appropriate analytical techniques and instrumentation required for quantifying VOCs.

Many technical papers discuss a wide range of organic pollutants and their levels in occupational and domestic environments. The crucial question is just what levels are "typical". Variations in outdoor concentrations, sampling and analytical techniques, and a lack of standardised experimental methods, makes comparisons between research results difficult, but not impossible to accomplish.

4.2 The Origin of Volatile Organic Compounds

Organic compounds can be characterised as compounds containing carbon and one or more other elements, most often hydrogen, oxygen, nitrogen, sulphur or other halogens (Maroni *et al.* 1995). A large fraction of organic compounds melt below 300 ° C, although exceptions exist.

According to Merian (1981), coal tar was once the only source of aromatic and some heterocyclic compounds. Petroleum was the source of aliphatic compounds that contain such substances as gasoline, kerosene, and lubricating oil. Natural gas contains methane together with smaller amounts of ethane, ethylene, benzene, toluene, cyclopentadine, naphthalene and nonhydrocarbon products as hydrogen, ammonia, carbon monoxide, carbon dioxide, hydrogen sulphide, hydrogen cyanide and nitric oxide. These three categories of natural substances are

still the major sources of organic compounds for most countries. Where petroleum is not available, a synthetic organic chemical industry can be based on acetylene, which in turn can be synthesised from limestone and coal.

Organic compounds generally dissolve in nonpolar solvents (liquids without localised electrical charges) e.g. carbon tetrachloride, or solvents of low polarity such as alcohols, acetic acid and acetone (Encyclopedia Britannica 1994). Organic solvents are often insoluble in water therefore they evaporate and stay in the atmosphere for quite some time before degrading (Korte & Bodefeld 1978, Merian 1981).

Hydrocarbons are a family of organic compounds composed entirely of carbon and hydrogen. They are the organic compounds of simplest composition and may be considered the parent substances from which all other organic compounds are derived (Encyclopedia Britannica 1994).

The hydrocarbons are conveniently classified into two major groups, open chain and cyclic. In open-chain compounds, containing more than one carbon atom, the carbon atoms are attached to each other to form a chain which may carry one or more side branches (Brady & Humiston 1986). In cyclic compounds the carbon atoms form one or more closed rings. The two major groups are subdivided according to chemical behavior in saturated and unsaturated compounds. The term aromatic hydrocarbon was originally restricted to the coal tar derivative benzene and its derivatives, but it now includes about half of all organic compounds, the remaining half being classified as aliphatic (Morrison & Boyd 1983). Overall, the occurrence and sources of hydrocarbons in the indoor environment, particularly those of the aromatic/aliphatic class, are extensive when compared to other organic and inorganic compounds. Therefore aromatic/aliphatic hydrocarbons are the focus of this Tasmanian study.

4.3 Physico-Chemical Properties

4.3.1 SATURATED OPEN-CHAIN HYDROCARBONS - ALIPHATIC HYDROCARBONS (ALKANES)

The saturated open-chain hydrocarbons form a homologous series called paraffin series or the alkane series (Sykes 1986). The composition of each of the series corresponds to the general formula C_nH_{2n+2}. Among the members of the series are methane (CH_4), ethane (C_2H_6), propane (C_3H_8) and butane (C_4H_{10}) (Fluka Chemie 1997/1998).

All members of the series are unreactive, for they do not react at ordinary temperatures with such reagents as acids, alkalies or oxidisers. The first four

members of the series are gases at ordinary temperatures and pressures, intermediate members are liquids, and the heavier members are solids or semisolids. Indoor sources of alkanes are petroleum products, gasoline, kerosene, lubricating oils, petroleum jelly, and paraffin.

Some alkanes have isomers that are found in indoor air, these are *n*-butane, isobutane, *n*-octane, *n*-nonane, and *n*-decane (Amdur *et al.* 1991, Maroni *et al.* 1995). Specific indoor sources of alkanes are illustrated in table 4.1.

4.3.2 UNSATURATED OPEN CHAIN HYDROCARBONS - ALIPHATIC HYDROCARBONS (ALKENES)

The unsaturated open-chain hydrocarbons include alkene or olefin series, the diene series, and the alkynene series. The alkene series is made up of chain hydrocarbons in which a double bond exists between two carbon atoms (Morrison & Boyd 1983). The general formula for the series is C_nH_2n. As in the paraffin series, the lower members are gases, intermediate compounds are liquids, and the higher members of the series are solids. The alkene series compounds are more active chemically than the saturated compounds, and easily react with substances such as halogens by adding atoms at the double bonds (Amdur *et al.* 1991). They are not found to a great extent in natural products, except in plant emissions e.g. pine and eucalyptus (isoprene and pinene), but are produced in the destructive distillation of complex natural substances such as coal, and are formed in large amounts in petroleum refining, particularly in the cracking process (Merian 1981). Some simple alkenes are ethene, propene, butene and pentene. Specific indoor sources of alkenes are illustrated in table 4.1.

4.3.3 UNSATURATED OPEN CHAIN HYDROCARBONS - ALIPHATIC HYDROCARBONS (ALKYNES)

The members of the alkyne group contain a triple bond between carbon atoms in the molecule atoms (Morrison & Boyd 1983). They are very active chemically and are not found free in nature. Their general formula is CnH_2n-_2, and contain still fewer hydrogen atoms than alkanes or alkenes (Cahn & Dermer 1979). They form a series analogous to the alkene series, the first of which is ethyne, followed by propyne, butyne and pentyne. Specific indoor sources of alkynes are illustrated in table 4.1.

TABLE 4.1

Indoor sources of aliphatic hydrocarbons
(compounds shown in parentheses indicate specific contaminants)
(Source: Adapted from Maroni *et al.* 1995, p35-38)

	Aliphatic hydrocarbons		
Indoor source	**Alkanes**	**Alkenes**	**Alkynes**
Consumer and commercial products	*(n-decane and branched alkanes)*		
Paints and associated products	*(n-hexane and n-heptane)*		
Pesticide products	*(kerosene)*		
Adhesives	*(hexane and heptane)*		
Automotive products	*(kerosene and mineral spirits)*		
Building materials	*(n-decane and n-dodecane)*		
HVAC systems			*(furnaces)*
Combustion products	*(butane and isobutane)*		
Biological contamination	*methane*		
Outdoor air			

Legend

Present in indoor air

Absent in indoor air

4.3.4 SATURATED CYCLIC HYDROCABONS

The simplest of the saturated cyclic hydrocarbons or cycloalkanes is cyclopropane (C_3H_6), the molecules of which are made up of three carbon atoms to each of which two hydrogen atoms are attached (Sykes 1986). Cyclopropane is somewhat more reactive than the corresponding open-chain alkane, propane. Other cycloalkanes make up part of ordinary gasoline and other combustion products commonly found in indoor air. Specific sources of saturated cyclic hydrocarbons are illustrated in table 4.2.

4.3.5 UNSATURATED CYCLIC HYDROCARBONS (AROMATICS)

Several unsaturated cyclic hydrocarbons, having the general formula $C_{10}H_{16}$, occur in certain fragrant natural oils that are distilled from plant materials. These hydrocarbons are terpenes and include α-pinene, β-pinene in turpentine, and limonene, in lemon and orange oils (Morrison & Boyd 1983, Sykes 1986).

The most important group of unsaturated cyclic hydrocarbons found in indoor air are the aromatics, which occur in coal tar. Although the aromatics sometimes exhibit unsaturation, with the addition of other substances, the principle reactions bring about the replacement of hydrogen atoms by other kinds of atoms or groups of atoms. The aromatic hydrocarbons include benzene (C_6H_6), ethylbenzene (C_8H_{10}), toluene (C_7H_8), *m-,p-,o*-xylene, 1,2,4-trimethylbenzene (C_9H_{12}), styrene (C_8H_8), and napthalene (Snyder 1987). Benzene and toluene come from paints, varnishes, glues, inks, enamels, lacquers and cleaning fluids. Xylenes ($C_6H_4(CH_3)_2$) and their three isomers, *m-*, *p-*, *o-*, are commonly found in solvents, cleaning agents and degreasers.

Aromatics usually contain closed rings of carbon atoms, but some aromatic rings may also contain an oxygen or nitrogen atom (Sykes 1986). Certain derivatives of aromatic compounds have special properties and special names, for example the addition of the hydroxyl group to an aromatic hydrocarbon yields a phenol (C_6H_6O) (hydroxy benzene). Common commercial applications indoors include disinfectants, extractive solvents, and wood preservatives. Specific indoor sources of aromatic hydrocarbons are illustrated in table 4.2.

4.3.6 HALOGENATED HYDROCARBONS

Halogenated hydrocarbons or halocarbons, is a chemical compound made up of the element carbon and one or more of the halogens bromine, chlorine, fluorine and iodine (Encyclopedia Britannica 1994).

TABLE 4.2

Indoor sources of saturated/unsaturated cyclic and halogenated hydrocarbons
(compounds shown in parentheses indicate specific contaminants)
(Source: Adapted from Maroni *et al.* 1995, p35-38)

	Saturated/unsaturated cyclic and halogenated hydrocarbons		
Indoor source	**Saturated cyclic**	**Aromatics**	**Halogenated**
Consumer and commercial products		*(toluene and xylene)*	*(1,1,1 -trichloroethane, tetrachloroethene)*
Paints and associated products		*(toluene)*	*(methylene chloride and propylene dichloride)*
Pesticide products		*(xylene)*	*(chlordane, diazinon, and 1,4-dichlorobenzene)*
Adhesives			
Automotive products		*(benzene, toluene and xylene)*	*(tetrachloroethene)*
Building materials		*(benzene, toluene, styrene and ethylbenzene)*	*(vinyl chloride)*
HVAC systems	*(furnaces)*		
Combustion products			
Biological contamination		*(toluene)*	
Outdoor air			

Legend

Present in indoor air

Absent in indoor air

There are two important subclasses of halocarbons, those of the chlorocarbon class and the fluorocarbons. Examples of chlorocarbons are carbon tetrachloride and tetrachloroethylene (Amdur 1991). Of all the halocarbons 1,1,1-trichloroethane ($C_2H_3CI_3$ (CI_3CCH_3)), is one of the most recognised in indoor air (Maroni *et al.* 1995). Specific indoor sources of halogenated hydrocarbons are illustrated in table 4.2.

4.3.7 OXYGENATED HYDROCARBONS (ALCOHOLS)

Alcohols are a class of organic compounds containing the hydroxy group OH, attached to a carbon atom. Alcohols have one, two, or three hyroxyl groups attached to their molecules are classified as monohydric, dihydric or trihydric. Methanol (CH_3OH) and ethanol (C_2H_6O) are monohydric alcohols (Morrison & Boyd 1983). Alcohols are further classified as primary, secondary or tertiary, depending on whether one, two or three other carbon atoms are bound to the carbon atom to which the hydroxyl group is bound (Cahn & Dermer 1979). Alcohols, although analogous to inorganic bases, are neither acid nor alkaline.

Higher alcohols, greater than ethyl alcohol, are isopropyl alcohol (which is commonly used indoors) butyl alcohol, and glycerin (Amdur 1991).

Alcohols like ethanol and methanol come from window cleaners, paints, paint thinners, and adhesives (Maroni *et al.* 1995). Specific indoor sources of alcohols are illustrated in table 4.3.

4.3.8 OXYGENATED HYDROCARBONS (KETONES)

Ketones are a class of organic compounds of the general structure R-CO-R', in which the R and R' represent organic radicals (Morrison & Boyd 1983, Sykes 1986). The simplest known ketone is acetone (C_3H_6O). Acetone is a product of the metabolism of fats, but under ordinary conditions it oxidises quickly to water and carbon dioxide (Amdur *et al.* 1991). Aside from acetone, ethylmethyl ketone ($C_2H_5COCH_3$) and dimehtyl ketone are the most common indoors (Maroni *et al.* 1995). Specific indoor sources of alcohols are illustrated in table 4.3.

4.3.9 OXYGENATED HYDROCARBONS (ALDEHYDES)

Aldehydes have the general formula; R-CH=O, where R is either a hydrogen atom, as in the case of formaldehyde, or an aliphatic or an aromatic hydrocarbon group. Formaldehyde (HCHO), is the simplest of the aldehydes. At ordinary temperatures it is a gas with a very pungent odour (Sykes 1986).

TABLE 4.3

Indoor sources of alcohols, ketones and aldehydes
(compounds shown in parentheses indicate specific contaminants)
(Source: Adapted from Maroni *et al.* 1995, p35-38)

	Oxygenated hydrocarbons		
Indoor source	**Alcohols**	**Ketones**	**Aldehydes**
Consumer and commercial products		*(acetone and methyl ethyl ketone)*	*(formaldehyde)*
Paints and associated products			
Pesticide products		*(methyl isobutyl ketone)*	
Adhesives		*(acetone)*	*(formaldehyde)*
Automotive products	*(isopropyl alcohol and ethylene glycol)*		
Building materials		*(acetone)*	*(formaldehyde)*
HVAC systems			
Combustion products			*(formaldehyde and acetaldehyde)*
Biological contamination	*(3-methyl-1-butanol)*	*(acetone)*	*(acetaldehyde)*
Personal products	*(isopropyl alcohol)*		
Outdoor air			

Legend

Present in indoor air

Absent in indoor air

Other aldehydes include acetanal (acetaldehyde) (CH_3CHO), butanal, hexanal, and nonanal (Cahn & Dermer 1979). It is employed as a disinfectant (formalin), insecticide, and fungicide. It is extensively used in the chemical industry in the synthesis of organic compounds (Maroni *et al.* 1995). Its most important use is in the manufacture of synthetic resins.

There are numerous indoor sources of formaldehyde include cleaning fluids, paints, fire retardents, adhesives, carpet backings, tobacco, naturals gas, particle board, disinfectants, varnishes, and insulation. Specific indoor sources of alcohols are illustrated in table 4.3.

4.3.10 OXYGENATED HYDROCARBONS (ESTERS)

Esters are compounds formed by the reaction of acids and alcohols, with the elimination of water. The esters of organic acids are usually colourless neutral liquids and generally insoluble in water but readily soluble in organic solvents (Encyclopedia Britannica 1994). Esters such as amyl acetate (banana oil), ethyl acetate, and cyclohexanol acetate are the principal solvents for lacquer preparations and are commonly found in indoor air. Indoor sources of esters are illustrated in table 4.4.

4.3.11 OXYGENATED HYDROCARBONS (ETHERS)

Esters are closely related to alcohols and prepared directly from them. The chemical formula for ether is R-O-R', in which O is an oxygen atom and R and R' represent either the same of different organic radicals itself (Sykes 1986). Chemically, ethers are inert, stable compounds, not readily affected by alkalies or acids. Most ethers are volatile, light flammable liquids, soluble in alcohols and other organic solvents. Examples of ethers used in indoor environments are methoxymethane, methyl ether, ethylene glycol ether, and dimethyl ether (Maroni *et al.* 1995). Indoor sources of ethers are illustrated in table 4.4.

4.3.12 OXYGENATED HYDROCARBONS (ACIDS)

Acids are defined by two groups, these are carboxylic acids and sulfonic acids. Carboxylic acid molecules are organic compounds containing one two or several carboxyl groups, and is the class generally found (Sykes 1986). Many acids are major industrial chemicals the most recognisable being acetic acid (CH_3COOH) used in the preparation of acetate, rayon, and paint solvents (Amdur 1991). Indoor sources of acids are illustrated in table 4.4.

TABLE 4.4

Indoor sources of esters, ethers and acids
(compounds shown in parentheses indicate specific contaminants)
(Source: Adapted from Maroni *et al.* 1995, p35-38)

	Oxygenated hydrocarbons		
Indoor source	**Esters**	**Ethers**	**Acids**
Consumer and commercial products	*(alkyl ethoxylate)*	*(glycol ethers)*	
Paints and associated products	*(ethyl acetate)*	*(methyl ether)*	
Pesticide products			
Adhesives	*(vinyl acetate)*		
Automotive products			
Building materials	*(urethane and ethyl acetate)*		
HVAC systems			
Combustion products			
Biological contamination			
Personal products			
Outdoor air			

Legend

Present in indoor air

Absent in indoor air

4.4 Typical Concentrations

A number of studies undertaken on indoor VOCs conclude that generally concentrations exceed those found outside, but are usually well below current permissible exposure limits. VOCs found in office air and other non-industrial building spaces vary considerably relative to types of compounds and concentrations of individual VOCs assessed as TVOCs.

Published frequency distributions of VOC concentrations in the literature, reveal a general consensus of study outcomes. It has been concluded that despite different objectives and protocols there is a reasonable agreement on the results characterised (WHO 1989). This is illustrated in table 4.5, which outlines the combined results of early VOC field studies. According to Maroni *et al.* (1995), the World Health Organisation's findings derived from these studies are representative of normal indoor situations and useful for estimating population exposures.

By reviewing past and current studies, a clear pattern emerges on the types of VOCs detected, even though limited data exists, and not all types of compounds are reported. VOCs of the aliphatic and aromatic class appear to be the most dominant, followed by alcohols, ketones, polyaromatic hydrocarbons and aldehydes. Maroni *et al.* (1995) state that in recent years VOCs of a more polar character such as glycol ethers and esters are being increasingly detected indoors. Many new consumer products and paints are believed to be responsible for this shift in prevailing compounds.

Table 4.6, summarises the VOC investigations undertaken and the subsequent levels detected. However, this table does not indicate, in general, whether certain types of compounds are being found more often than others. This is an issue that needs to be considered in further VOC studies. Of the compounds listed from the 23 studies, compounds most frequently identified were toluene, benzene, xylenes and formaldehyde. Other studies where specific sampling and analysis were performed have also reported similar results.

There are literally hundreds of VOCs in indoor air, and studies into their concentrations have identified merely a portion of the chemical compounds present. There is a wide variety yet unidentified, including the compounds resulting from combined effects.

TABLE 4.5

Concentrations and distributions of organic compounds in indoor air
(Source: Maroni *et al.* 1995, p49-52)

Pollutant	Category	Concentration ($\mu g/m^3$) Percentile					Sources
		10th	50th	90th	98th	Outdoor	
Aliphatic hydrocarbons							
n-hexane	2	4	10	20			solvent, fuel component
n-heptane	2	3	5	15	60		solvent, fuel component
n-octane	2	2	5	10			solvent, fuel component
n-decane	2	3	10	50	90		solvent, fuel component
n-undecane	2	3	5	25			solvent, fuel component
n-dodecane	2	2	5	10			solvent, fuel component
n-tridecane	2	2	5	10		<0.3	solvent, fuel component
n-tetradecane	2		2	10		<0.3	solvent, fuel component
n-pentadecane	2-3		1	5		<0.3	solvent, fuel component
n-hexadecane	2-3		1	5		<0.3	solvent, fuel component
2-methylhexane	2		5	100		2	solvent, fuel component
3-methylhexane	2		2	100		2	solvent, fuel component
Terpenes							
limonene	2	2	15	70			odourant, detergent
α-pinene	2	2	10	20			wax, wood product
β-pinene	2	<1	<1	5			wax, wood product
α-terpinene	2	1	5	10			wax, wood product
Esters and ketones							
ethylacetate	2						solvent
n-butyl acetate	2						solvent
ethylmethyl ketone	2	<2	5	10			solvent
4-methyl-2-pentanone	2		2				solvent

Category 1 - Very volatile organic compounds (VVOC)
Category 2 - Volatile organic compound (VOC)
Category 3 - Semivolatile organic compound (SVOC)

TABLE 4.5 Continued

Concentrations and distributions of organic compounds in indoor air
(Source: Maroni *et al.* 1995, p49-52)

Pollutant	Category	Concentration ($\mu g/m^3$) Percentile					Sources
		10th	50th	90th	98th	Outdoor	
Chlorinated hydrocarbons							
chloroform	1-2			3	15		drinking water
dichloromethane	2			<10	<10		aerosol, paint remover
tetrachloromethane	2		1	20			industrial solvent
bromoform	2						drinking water
1,2-dichloroethane	2						cleaning agent, additive
1,1,1-trichloroethane	2	2	5	20			dry cleaning solvent
trichloroethylene	2	1	5	20	30	<2	solvent, spot remover
tetrachloroethylene	2	2	5	20	70		dry cleaning solvent
chlorobenzene	2		<0.5	10			solvent, textile additive
m-dichlorobenzene	2		<0.5	5		<0.5	deodorant, moth balls
p-dichlorobenzene	2	1	5	20			deodorant, moth balls
1,2,3-trichlorobenzene	2		1	10		<1	dye carrier, pesticide
1,2,4-trichlorobenzene	2		1	15		<1	dye carrier, pesticide
1,3,5-trichlorobenzene	2			5		<1	dye carrier, pesticide
Alcohols							
ethanol	1						solvent, beverages, fuel
propanol	1-2						solvent
n-butanol	2	<1	<1	3			solvent
isobutanol	2	<1	1	5			solvent
pentanol	2						solvent, plasticiser
hexanol	2						solvent, plasticiser
2-ethylhexanol	2	<1	1	5			solvent, plasticiser
Other compounds							
Napthalene	3		2	5			solvent, moth balls

TABLE 4.5 Continued

Concentrations and distributions of organic compounds in indoor air
(Source: Maroni *et al.* 1995, p49-52)

Pollutant	Category	Concentration ($\mu g/m^3$) Percentile					Sources
		10th	50th	90th	98th	Outdoor	
Aromatic hydrocarbons							
benzene	2	2	10	20	30	3	fuel component
toluene	2	30	65	150	250	5	fuel component
m,p-xylene	2	10	20	40		2	fuel component solvent
o-xylene	2	3	5	10		1	fuel component solvent
ethylbenzene	2	4	10	20	10	1	fuel, solvent
n-propylbenzene	2	<1	2	6		<0.3	fuel, solvent
isopropylbenzene	2		1	3		<0.3	fuel, solvent
o-methylethylbenzene	2		2	5		<0.3	fuel, solvent
1,2,3-trimethylbenzene	2		2	5		<0.3	fuel, solvent
1,2,4-trimethylbenzene	2	5	20			1	fuel, solvent
1,3,5-trimethylbenzene	2	2	5			<0.3	fuel, solvent
n-butylbenzene	2		1	10		<0.3	fuel, solvent
p-methylisopropyl-benzene	2		1	10		<0.3	fuel, solvent
diethylbenzene	2						fuel, solvent
styrene	2	<1	1	5	10		fuel component
Aldehydes							
formaldehyde			25	60			chipboard, insulation
acetaldehyde			10	30			cigarette smoke
acrolein							cigarette smoke
butanal			1	5			
hexanal	2	<1	1	5			paper, paints
nonanal	2						paper, paints, flavour

TABLE 4.6

Summary of published indoor VOC investigations

AUTHOR	YEAR	POLLUTANT	LEVELS
Snyder	1987	Benzene	10 ppm
		Toluene	200 ppm
		Xylene	100 ppm
Hedge *et al.*	1989	TVOC	8.9-86.6 mg/m^3
De Bortoli *et al.*	1990	TVOC	0.22- 3.93 mg/m^3
Skov *et al.*	1990	TVOC	0.43-2.36 mg/m^3
Valbjorn *et al.*	1990	Formaldehyde	0.1-0.4 ppm
Hodgson *et al.*	1991	TVOC	0.48-1.95 ppm
Liao *et al.*	1991	Benzene	179 µg/m^3
		Toluene	81 µg/m^3
Andersson *et al.*	1992	TVOC	100-700 µg/m^3
Haghighat *et al.*	1992	TVOC	700-9200 µg/m^3
Hodgson *et al.*	1992	TVOC	3.0-4.8 ppm
Broder *et al.*	1993	TVOC	150-49100 µg/m^3
Brown	1993	Formaldehyde	0-1300 ppb
Fisk *et al.*	1993	TVOC	340-1200 µg/m^3
Leaderer *et al.*	1993	Hydrocarbons	100-3000 ppm
Kostiainen *et al.*	1995	TVOC	40-235 µg/m^3
Nelson *et al.*	1995	TVOC	9-54.7 µg/m^3
Reiss *et al.*	1995	Organic acids	9.8 - 17.8 ppb
		Acetic acid	15.5 - 28.7 ppb
		Xylene	150 ppb
Brown	1996	TVOC	130-10000 µg/m^3
Vanosdell *et al.*	1996	VOC mix	0.5 - 100 ppm
Molhave *et al.*	1996	VOC mix	30 µg/m (2)/h
Crump *et al.*	1997	TVOC	57-1954 µg/m^3
Drahonovská &	1997	VOC mix	5 - 726 µg/m^3
Gajdos			23 - 758 µg/m^3
			5 - 426 µg/m^3
			16 - 238 µg/m^3
Santos *et al.*	1997	VOC mix	0.94-305.4 mg/m^3

4.5 Conclusion

Modern methods of scientific analysis has added to the understanding of the presence of organic compounds in the indoor environment. Indoor air field studies have been successful in determining the frequency distribution of measured concentrations of a multitude of VOCs which are representative of indoor environments. Although detailed information on organic compounds found in indoor air is still incomplete, VOC concentrations that have been identified and tentatively identified, are extremely useful in assessing population exposures.

CHAPTER FIVE

IAQ LEGISLATIONS, STANDARDS and GUIDELINES

5.1 Introduction

Indoor air pollution poses many challenges to the health and safety of occupants in non-industrial buildings. As awareness and concern for atmospheric hazards increase, pressure escalates on appropriate authorities to regulate all aspects of the internal environment, including non-industrial workplaces like offices.

Responsibility for health issues relating to indoor air quality has been multi disciplined, encompassing many government departments and committees. To date these fragmented, independent activities, are proving unsatisfactory because problems related to the indoor environment are on the increase. Therefore methods of regulatory control currently used appear to be having little or no effect on improving indoor air. This is especially the case for volatile organic compound control which has proven to be beyond conventional regulatory mechanisms.

Overall, the formulation and implementation of suitable standards and guidelines pertaining to VOCs in indoor air is an area which has been largely neglected, especially in Australia. In some cases it is necessary to refer to information from other countries, or to publications that have a close association with indoor air or VOCs e.g. outdoor air quality standards or exposure standards for atmospheric contaminants in the occupational environment. Regardless of this difficulty, the information currently available on standards and guidelines are valuable when determining if measured concentrations of pollutants are at levels considered acceptable or hazardous (such as the VOCs examined in this Tasmanian study on office buildings). In addition, an outline of current standards and guidelines provides a reference for evaluating what control measures are available for office environments, and their usefulness in gauging the relative performance of the indoor environment.

5.2 The Legal Aspects of Indoor Air Quality

To date, there have been very few laws developed specifically for indoor air (non-industrial) environments in any country. According to Maroni *et al.* (1995), many countries have regulations which limit emissions of air pollutants, but in the majority of cases these are applicable to outdoor ambient air rather than indoor air (industrial/non-industrial). This is seen in table 5.1, which summarises the standards and guidelines available in Australia for various categories of air pollution. Air in working environments has been subject to regulations but generally these are defined as short term exposure limits (STEL), time weighted averages (TWA) and peak limitations. More often than not these regulations are

inappropriate for the low concentrations of VOCs present in indoor air, therefore the non-industrial indoor environment remains without protective measures.

TABLE 5.1

Summary of standards, guidelines and research activity in Australia

Category	Standards available	Guidelines available	Research activity
Outdoor ambient air	O	O	O
Indoor air (industrial workplace)	O	O	O
Indoor air (non-industrial workplace)	O	O	O
Private homes	O		O

Legend

Adequate information exists. Standards and guidelines are available for this category.

Adequate information exists. Standards and guidelines are available for this category.

Limited information exists. Standards and guidelines used are taken from outdoor air and industrial workplace air. Specific standards and guidelines for non-industrial workplaces are only available for human thermal comfort, ventilation, lighting, and noise.

Almost no information exists. Specific standards for private homes are those outlined in the Australian Building Code (1994), and apply to the construction of buildings.

Brown (1997) extends on this point by stating that there are several additional reasons for the absence of legislation and regulatory implementation, these are:

1. Private indoor environments such as residences are regarded by the public as sacrosanct, requiring minimum imposition of regulations;
2. Enforcement of regulation in residences would be impossible due to their large numbers;

3. No single government authority has responsibility for indoor air quality; and
4. Indoor air quality involves a complex set of factors (e.g. building and ventilation system design, construction, operation and maintenance, outdoor climate/pollutant sources, a diverse range and mixture of pollutants, multiple indoor pollutant sources, diverse health effects).

At present, few regulatory models limiting indoor sources and emissions are found throughout the world, particularly those pertaining to office air. Given the complexity of indoor environments it is likely that any regulations will not be easily established. Inspite of the difficulties, there are possibilities of achieving an acceptable indoor air quality, and some activities currently seen elsewhere are promising. Overall, because information is so scarce on controlling indoor contaminants, it is usually necessary to draw on information from many countries.

5.3 International Legislations, Standards and Guidelines

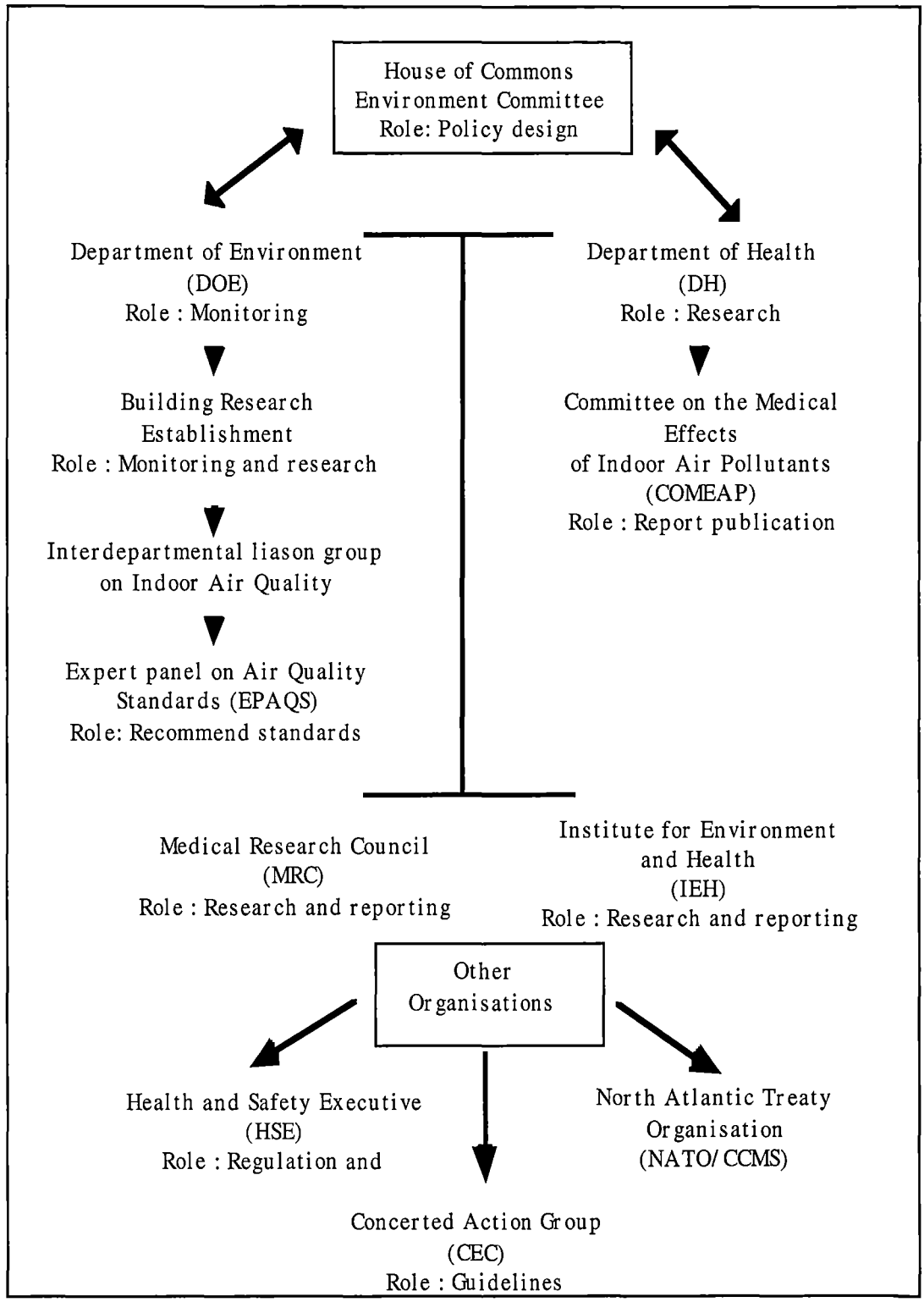
5.3.1 ENGLAND

According to Harrison (1996), management and assessment of air quality (primarily for outdoor air, but their relevance to indoor air has recently become of interest (Harrison 1996)), involve a collaboration of many departments in England, as seen in figure 5.1. The Department of Environment (DOE) takes the primary role in sponsoring other organisations like the Building Research Establishment (BRE), to undertake monitoring work on base line pollution levels on a range of indoor environments, including private dwellings. Results found in the DOE and BRE reports are then submitted to the House of Commons Environment Committee who have the task of relaying the findings to the Government, which in turn sets appropriate laws (DOE 1990, DOE 1995a).

Harrison (1996) states that even though the EPAQS (Expert Panel on Air Quality Standards who are responsible for establishing standards) recommends standards for outdoor air it acknowledges possible indoor exposures where significant. At present, England is at the early stages of developing IAQ regulations (DOE 1995a, DOE 1995b). In an attempt to achieve improvement in indoor environments, guidance notes have already been published (DOE 1990, DOE 1995c), reports on health effects (IEH 1995), as well as other measures including future voluntary regulations on emissions and dissemination of public information. This has already been undertaken by the DOE in conjunction with the UK's National Environmental Health Action Plan to complement outdoor air quality

FIGURE 5.1

Departments involved in the control of indoor air pollution in England



strategies. England, like many other countries, is still assessing the risks to health resulting from indoor exposures, therefore it will be quite some time before regulations will be formulated.

5.3.2 THE UNITED STATES OF AMERICA

The most recognisable American group to be associated with indoor air quality is the American Conference of Governmental Industrial Hygienists (ACGIH) (figure 5.2). The ACGIH focus primarily on research, development of guidelines, and standards. They annually publish a list of acceptable workplace exposure concentrations for over 500 toxic chemicals and dusts, including VOCs commonly found in indoor air.

The USEPA is similar in function to the ACGIH, and issues a collection of national standards but these are for outdoor ambient air (under The Clean Air Act of 1970, revised in 1971 and 1989), although the USEPA has set several standards for VOCs that can enter the air through volatilisation from water used in a residence or other building. The publications from both the ACGIH and USEPA are recognised and used globally (American Lung Association *et al.* 1994, Maroni *et al.* 1995, NOHSC:1003 1995, Sigma-Aldrich 1996). In Australia, ACGIH and USEPA publications are extensively used for hazard identification and exposure standards (occupational environment) (PCA 1994).

Most health standards relating to indoor air in workplaces (derived from analysis of dose-response relationships) are established by the Occupational Safety and Health Administration (OSHA) (Turiel 1985). Unfortunately, these standards are only applicable to industrial environments, neglecting non-industrial settings.

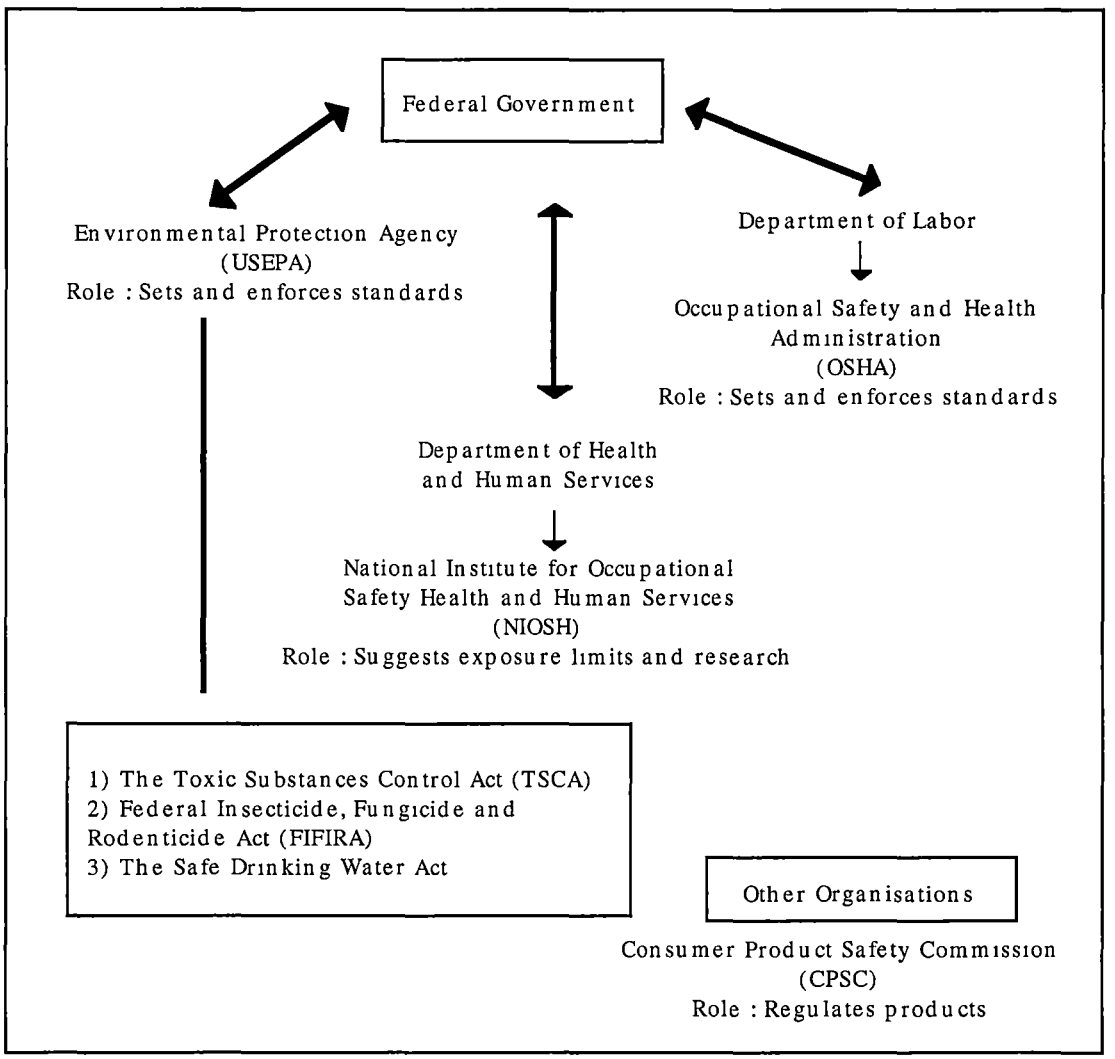
The National Institute of Occupational Safety and Health (NIOSH), a research arm of OSHA, have been modifying OSHA industrial standards to suit a long category of workers whose activities are not carried out in an industrial environment. According to the American Lung Association *et al.* (1994), NIOSH is generally involved in a whole range of activities including research, planning, directing, recommending occupational health and safety standards, and ensuring that there is compliance with protocol.

In addition, other groups such as the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE), establish standards and guidelines for indoor environments, but these are typically invoked only for new or renovated construction. ASHRAE standards, like ACGIH and USEPA standards, are used as a benchmark in countries all over the world. For example, in Australia, ASHRAE standards and guidelines are used to manage indoor air quality (including offices) focusing on factors such as human thermal comfort (ASHRAE 55-1981), ventilation (ASHRAE 62-1989), pollutants, lighting and noise (PCA 1994). The Consumer Product Safety Commission (CPSC) also has a small role by regulating products which may release indoor air pollutants.

Overall, there are a range of standards and guidelines for non-industrial indoor environments in the United States, including source emission standards that specify maximum rates at which contaminants can be released from a source.

FIGURE 5.2

Departments involved in the control of the indoor environment in America



5.3.3 THE NORDIC COUNTRIES

One of the most recognised Nordic groups associated with non-industrial indoor air quality control is the Nordic Committee on Building Regulations (NKB) (Maroni *et al.* 1995). Maroni *et al.* (1995) state that the NKB report (NKB No 41 "indoor climate"), published in 1981, has served as a basis for many national regulations in various countries. The NKB is recognised for collaborating with other bodies such as WHO and USEPA, particularly in activities concerning the formulation of guidelines and recommendations. Any guidelines set by the Nordic Committee on Building Regulations are used extensively in indoor air quality programmes elsewhere (USEPA) (Maroni *et al.* 1995).

According to Valbjorn *et al.* (1990), groups such as The Institute of Public Health, The Vardal Foundation, The Nordic Ventilation Group, The National Board

of Health and Welfare, The Danish Building Research Institute, and the National Board of Housing, Building and Planning have been actively involved in the preparation of guides for the indoor environment. Interestingly, research and subsequent publications issued by these groups focus almost exclusively on office environments.

5.3.4 CENTRAL AND EASTERN EUROPE

Political changes in central and eastern Europe have initiated new legislative organisational frameworks in environmental air quality. At present countries like Lithuania, Romania and the Ukraine are in the process of moving away from basic occupational health and safety regulations formulated by the former USSR.

Bitkolov and Musijchuk (1997) state that Russia has a range of standards for potentially dangerous chemical and biological substances in working zone air. Some of the standards apply only to companies or to the air quality of professional workers with radioactive materials (statutes NRB-76/87 and NRB-96) (Bitkolov & Musijchuk 1997), while the remaining standards include offices and domestic habitations. These are:

1. The temporary instruction for substantiation of the maximum allowable concentration (MAC) of pollutants in atmospheric air (1989); and
2. The lists and codes of pollutant substances (1995).

According to Bitkolov and Musijchuk (1997), Russia has a strict system of authorising chemicals for use in working zone air. More than 2000 MAC values have been officially adopted, but none apply exclusively to the low concentrations or mixtures of chemicals found in indoor air. In addition, the available current Russian standards are temporary and require toxicological verification (Bitkolov & Musijchuk 1997).

Although at initial stages, central and eastern european countries have set their own relevant policy initiatives. For example in Lithuania, the Section for Hygienic Regulation at the Centre of Environmental Medicine (CEM) is preparing documents for the national hygienic standard on indoor air quality. These documents outline maximum permissible levels of pollutants, chemical and physical, in dwellings and public buildings (Seskauskas & Kubiliené 1997). The national regulatory limits for air pollutants in Romania have also been changed with many non-governmental organisations creating national standards based on their own epidemiological investigations (Nicoara & Mocsy 1997). Dobrovolsky (1997) states that the Ukraine has followed a similar framework by setting up a national body, incorporating the Institute of Health, the Institute of Common and

Community Hygeine and the Ministry of Health, to work out standards specifying the indoor environment (Dobrovolsky 1997).

Even though there are disadvantages in the legislative developments of these countries, they have at least attempted change in regard to indoor air quality standards.

5.3.5 GREATER EUROPE

If countries have no laws or equivalent guidelines of their own regarding control of indoor air pollution, standards and guidelines established by other organisations are adopted. Examples of these include the International Standards Organisation (ISO), North Atlantic Treaty Organisation's Committee on the Challenges of Modern Society (NATO/CCMS), European Concerted Action Group (ECA), Commission of European Communities (CEC), and the World Health Organisation (WHO) (appendix 1). In Australia, publications and standards by WHO and the ISO are most frequently cited. For example, the Property Council of Australia (1994) suggest building owners and managers (in offices) use the ISO standard for acceptable factors concerning human thermal comfort (ISO-7730).

5.4 Australian Legislation, Standards and Guidelines

5.4.1 NATIONAL BODIES ASSOCIATED WITH INDOOR AIR

Indoor environment regulations in Australia are predominantly the responsibility of specific Federal and State government departments (appendix 2), with many having specific acts pertaining to indoor (industrial/non-industrial) environments.

The National Occupational Health and Safety Commission, National Occupational Health and Safety Office (NOHSO) and the National Institute of Occupational Health and Safety is a tripartite body established by the Commonwealth Government (figure 5.3). They are recognised as the leading organisation declaring foregoing principles in national standards and codes of practice (NOHSC 1003:1995). According to the NOHSC (NOHSC 1003:1995), the main purpose of the organisation is;

1. To develop among the members of the community an awareness of issues relevant to occupational health and safety matters and the facilitation of public debate and discussion on such issues;
2. To provide, in the public interest, a forum by which representatives of the Government of the Commonwealth, the Governments of the States

and of employers and employees may consult together in, and participate in the development and formulation of policies and strategies relating to, occupational health and safety matters; and

3. To provide a national focus for activities relating to occupational health and safety matters.

The standards declared by the NOHSC, under the National Occupational Health and Safety Act 1985 are documents which prescribe preventative exposure limits to avert occupational deaths, injuries and diseases in workplaces. The main focus of this Act is to facilitate and encourage the implementation of:

1. The policies and strategies formulated by the commission;
2. To enforce the recommendations made by the commission with respect to the taking of action or the making or review of laws or awards; and
3. The setting of national standards and codes of practice.

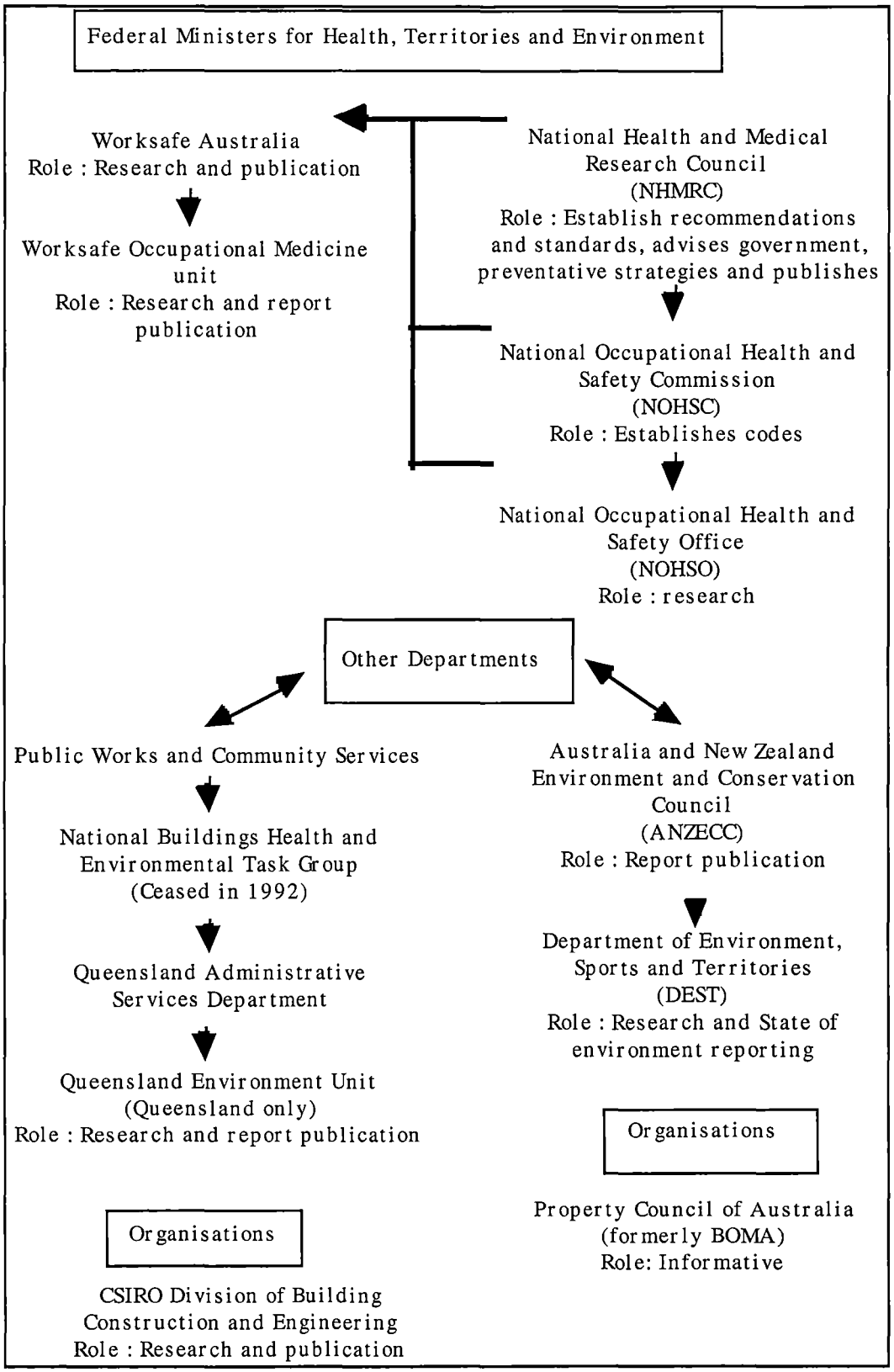
Unfortunately, there are few specific references in the NOHSC standards or guidelines pertaining to non-industrial workplaces, such as offices, but there are issues in the NOHSC occupational environment literature which are applicable to some non-industrial indoor air parameters. For example, there is a guidance note on passive smoking in the workplace (NOHSC 3019:1994), a guide on office copy machines (NOHSC:No 9027571), and a guide on legionnaires' disease and related illnesses (NOHSC:No 9027603).

The National Health and Medical Research Council, like the NOHSC has a substantial role in formulating recommendations by advising government in matters relating to health and research funding in the indoor air quality area. Similarly, Worksafe Australia (which is a subgroup of NOHSC) is dedicated to the research and publication of national standards and guidance notes. An example closely related to indoor air would be the publication on "exposure standards for atmospheric contaminants in the occupational environment" (NOHSC 1003:1995, NOHSC 3008:1995). Once again Worksafe's role is merely to inform the public of standards and guidelines, but they have no legislative power unless they are specifically incorporated into Commonwealth, State or Territory legislation (ANZECC 1994).

Interestingly, some states have developed their own Acts or research activities pertaining to indoor (industrial) environments. Victoria is one such example that has its own Occupational Health and Safety Act (1985) dealing with occupational environments, therefore Victoria does not rely solely on the NOHS Act, instead they use the two Acts simultaneously (ANZECC 1994).

FIGURE 5.3

Departments involved in the control of indoor air pollution in Australia.



Other departments and agencies who are associated with indoor air activities are the Department of Environment, Sport and Territories, environmental protection agencies, and the CSIRO Division of Building, Construction and Engineering. These bodies have a non-regulatory role and conduct either research or state of environment reporting. For example, the Victorian EPA (figure 5.4) has responsibility under the State Environment Protection Policy (The Air Environment) for only outdoor air, but has recently reviewed indoor air quality in residential buildings.

The Property Council of Australia (formerly known as the Building Owners and Managers Association (BOMA)), the Community Public Sector Union (CPSU) and Comcare (figure 5.4) have a programme of accommodation issues, which is illustrated in a publication called "Improving your work environment" (1996) (PCA 1994, CPSU & Comcare 1994, CPSU 1996). It covers many indoor air quality aspects including ventilation, pesticides and hazardous chemicals, and is targeted at union health representatives.

5.4.2 OTHER BODIES ASSOCIATED WITH INDOOR AIR

Local councils in all Australian states have no major function in controlling indoor air in non-industrial workplaces. Local councils merely reinforce the current Australian Building Code (ABC) (ABCB 1994) during and after completion of building works, with no subsequent follow up or monitoring of buildings. The Building Code covers issues like ventilation and fire escapes, but there is no mention of other areas of concern such as the type of materials to be used and placement of photocopiers in buildings such as offices.

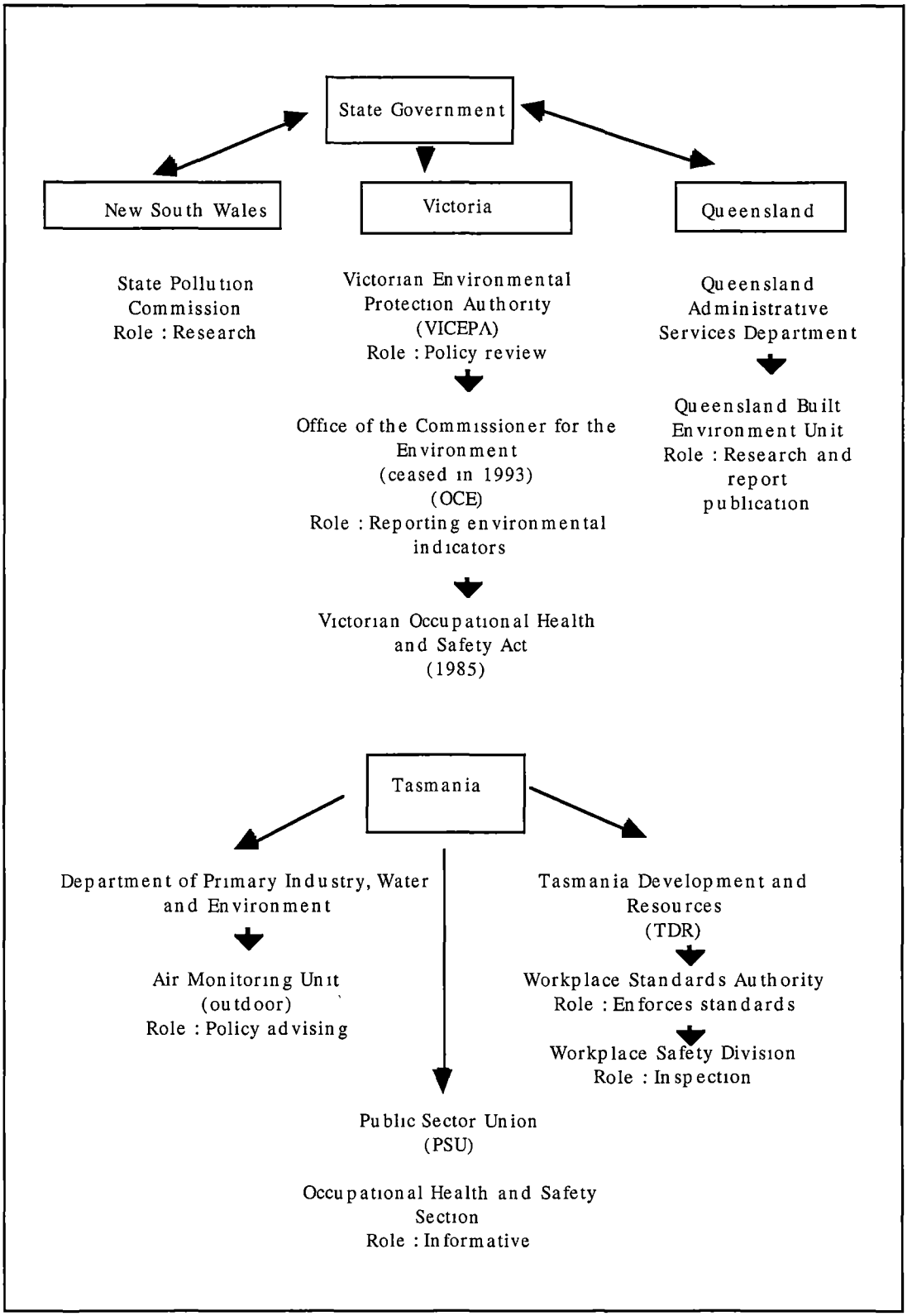
5.5 Tasmanian Legislation, Standards and Guidelines

The Workplace Standards Authority (WSA) is charged with the administration of the Workplace Health and Safety Act (1995), and its duties include achieving the best practice in workplace health and safety through ensuring compliance with legislations (figure 5.4).

Industrial hygienists from the WSA only examine indoor environments when a problem is drawn to their attention. Tasmania does not have a Clean Air Act like some other states, the only equivalent is the Environmental Management and Pollution Control Act (1994). Generally, indoor air issues (industrial/non-industrial) are dealt with in the National Occupational Health and Safety Act (1985) and the Public Health Act (1962) (ANZECC 1994).

FIGURE 5.4

Departments involved in the control of indoor air pollution in Australia at a State level



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There is also the Tasmanian Development Resources (TDR), who have a workplace safety division. Their role is primarily to advise current policies and to investigate air conditioning systems if there is a problem. The Department of Primary Industries, Water and Environment (DPIWE) and the Department of Health and Community Services have an air pollution section, yet their activities have very little relevance to formal non-industrial air quality investigations, instead they focus more on industrial work environments.

5.6 Legislation, Standards and Guidelines for VOCs

To date there exists no specific legislation, standards and guidelines for low level volatile organic compounds, but there are standards regarding higher concentrations of volatile organic compounds found in industrial settings.

Therefore, *"maximum allowable concentrations"* (MACs) workplace exposure limits are used as a control for VOC exposure. These MACs are commonly published as material safety data sheets and outline chemicals in the industrial environment. Material safety data sheets can be legislative, as in the case of OSHA who use a legal airborne permissible exposure limit (PEL), or a recommended exposure limit which is adpoted by organsiations like NIOSH and the ACGIH.

Material safety data sheets are employed as a guide to outline the maximum allowable concentrations in ppb (parts of vapour or gas per billion parts of contaminated air by volume), ppm (parts of vapour or gas per million parts of contaminated air by volume), or mg/m^3 (milligrams of substance per cubic metre of air at 25°C and one atmosphere pressure) (NOHSC 1995, ACGIH 1994). Compounds specified on these sheets are either on the hazardous substances list, regulated by OSHA and cited by ACGIH and USEPA, or on the special health hazard substance list.

In some countries, a different system of toxic categorisation is adopted for regualtory purposes (Ballantyne *et al.* 1995). The classification "LC 50" signifies inhalation limits to gases and vapours ($mg\ L^{-1}h^{-1}$), and is also used to determine the acute inhaled median lethal concentration. It essentially labels compounds as very toxic or toxic based on a system of inhalation assay studies. The acute systemic toxicity classification of LC 50 is only applied in Canada, Sweden, and the USA (OSHA), and not in Australia.

Similarly, WHO (1987) established air quality guideline values for VOCs, but as with other organisations, the primarily focus has been on outdoor rather than indoor air. But, in some instances, special attention has been given to contaminants encountered indoors (applicable to industrial/non-industrial). When guidelines are not available, risk estimates are used instead, as illustrated in table 5.2. This table

TABLE 5.2

Established guideline values and risk estimates for selected carcinogenic organic and inorganic substances in outdoor air
(Source: Maroni *et al.* 1995, p802)

Substance	IARC (group classification)	Risk estimate based on carcinogen endpoint	Guideline values based on: Toxicological endpoint	Sensory effects or annoyance	Ecological effects
Organic substances					
Acrylonitrile	2A	X			
Benzene	1	X			
Carbon disulfide		X	X		
1,2-Dichloromethane	a		X		
Dichloromethane	a		X		
Formaldehyde	2B		X		
Polynuclear aromatic hydrocarbons (benzo(a)pyrene)	b	X			
Styrene	3	X	X	X	
Tetrachloroethylene	3		X	X	
Toluene			X	X	
Trichloroethylene	3		X		
Vinyl chloride	1	X			
Inorganic substances					
Arsenic	1	X			
Asbestos	1	X			
Cadmium	2B		X		
Carbon monoxide		X			
Chromium (VI)	1	X			
Hyrogen sulfide		X	X		
Lead	3		X		
Manganese			X		
Mercury		X			
Nickel	2A[c]	X			
Nitrogen dioxide		X		X	
Ozone/photochemical oxidants			X		X
Radon			X		
Sulfur dioxide and particulate matter			X		X

a Not classified but sufficient evidence of carcinogenicity in experimental animals.

b Not classified, but sufficient evidence of carcinogencity of PAH in humans in some occupational exposures (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol.34). Sufficient evidence of carcinogenicity of benzo(a)pyrene is present as a component of the total content of PAHs in the environment (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 32).

c Exposures from nickel refineries are classified in Group 1.

TABLE 5.3

Guideline values for individual substances based on effects other than cancer or odour/annoyance in indoor air

(Source: Maroni *et al.* 1995, p804)

Substance	Time weighted average	Averaging time
Cadmium	1-5 ng/m^3	1 year (rural)
	10-20 ng/m^3	1 year (rural)
Carbon disulfide	100 µg/m^3	24 hr
Carbon monoxide	100 mg/m^3	15 min
	60 mg/m^3	30 min
	30 mg/m^3	1 hr
	10 mg/m^3	8 hr
1,2-Dichloroethane	0.7 mg/m^3	24 hr
Dichloromethane (methylene chloride)	3 mg/m^3	24 hr
Formaldehyde	100 µg/m^3	30 min
Hydrogen sulfide	150 µg/m^3	24 hr
Lead	0.5-1.0 µg/m^3	1 year
Manganese	1 µg/m^3	1 year
Mercury	1 µg/m^3	1 year
Nitrogen dioxide	400 µg/m^3	1 hr
	150 µg/m^3	24 hr
Ozone	150-200 µg/m^3	1 hr
	100-120 µg/m^3	8 hr
Styrene	800 µg/m^3	24 hr
Sulphur dioxide	500 µg/m^3	10 min
	350 µg/m^3	1 hr
Sulphuric acid		
Tetrachloroethylene	5 mg/m^3	24 hr
Toluene	8 mg/m^3	24 hr
Trichloroethylene	1 mg/m^3	24 hr
Vanadium	1 µg/m^3	24 hr

Exposure at these concentrations should be for no longer than the indicated times and should not be repeated within 8 hours.

Due to respiratory irritancy, it would be desirable to have a short-term guidelines, but the present data base does not permit such estimations.

The guidelines value is given only for indoor pollution, no guidance is given on outdoor concentrations (via deposition and entry into the food chain) that might be of indirect relevance.

NOTE: When air levels in the general environment are orders of magnitude lower than the guideline values, present exposures are unlikely to present a health concern. Guideline values in those cases are directed only to specific release episodes or specific indoor pollution problems.

details the established guideline values and risk estimates for some carcinogenic outdoor VOCs that are also detectable in indoor air. Table 5.3 further illustrates WHO guideline values for individual substances indoors based on effects other than cancer or odour annoyance.

The guidelines available are useful but neglect to include non-industrial buildings, like the offices sampled in this Tasmanian study. In addition, guideline values and standards do not take into consideration complex mixtures and low pollutant concentrations.

5.6.1 TENTATIVE STANDARDS FOR VOCs

Even though there is data on atmospheric contaminants and their acceptable levels, basic information is still missing on low level, synergistic pollutants. Control of indoor air quality requires the charactersisation of these to provide a basis for regulation.

The TVOC concept is one such tentative regulatory mechanism. Coined by Molhave and Nielsen (1992), the TVOC theory centres around establishing a dose-response relationship between discomfort and health effects and the level of volatile organic compounds present in the atmosphere. It is also seen as an indicator for sensory irritation or "provisional guideline" for acceptable ranges.

The TVOC concept works on the basis of summing several VOCs and analyses their total concentration. Molhave's original study (1986), examined a mixture of 22 non-carcinogenic VOCs which were representative of indoor air exposures. These were then used as a predictor of effects on human health, and as a possible basis for recommended allowable concentrations. The results, as illustrated in table 5.4, are a defined range of VOC concentrations and their subsequent health effects.

To obtain the TVOC sum, VOCs are ranked according to their measured concentration, and the concentrations of the first ten are added. As seen in table 5.5, this definition also allows a proposed target value to be set which is intended to represent normal indoor air conditions in non-industrial environments. According to Maroni *et al.* (1995), the target guideline for TVOC is proposed by Molhave (1995) to amount to 0.3 mg/m^3, with no individual VOC exceeding 50% of the concentration alloted to its class.

Following this new strategy in VOC evaluation, many countries are looking to embrace the guidelines based on this concept. In Australia, for example, the NHMRC has recommended an indoor air level of 500 $\mu g/m^3$ TVOC (one hour average), with no VOC exceeding 250 $\mu g/m^3$ (Brown 1993).

TABLE 5.4

Dose-response relationship between health effects and TVOC mixtures
(Source: Godish 1995, p155)

TVOC Concentration (mg/m^3)	Response	Exposure range
< 0.20	No effects range	Comfort range
0.20 - 3.0	Discomfort possible	Multifactorial exposure range
3.0 - 25.0	Irritation and discomfort	Discomfort range
> 25.0	Neurotoxic effects in addition to headaches	Toxic range

TABLE 5.5

Proposed target value for non-industrial buildings for TVOC
(Source: Molhave 1995)

Chemical class of VOC	Concentration ($\mu g/m^3$)
Alkenes	100
Aromatic hydrocarbons	50
Terpenes	30
Halocarbons	30
Esters	20
Aldehydes and Ketones	20
Other	50
Target guideline value (sum of VOC)	300

Many countries are still identifying ways of proposing strategies for defining TVOC and its guideline value. Aside from Norway who has set a level of 400 $\mu g/m^3$ for TVOCs (Maroni *et al.* 1995), other countries have set an alternative level of acceptable exposure ranges for indoor air pollutants by choosing to consider individual substances such as formaldehyde. Because of the great variety of VOCs and the difficulty in implementing standards, many countries are waiting until more

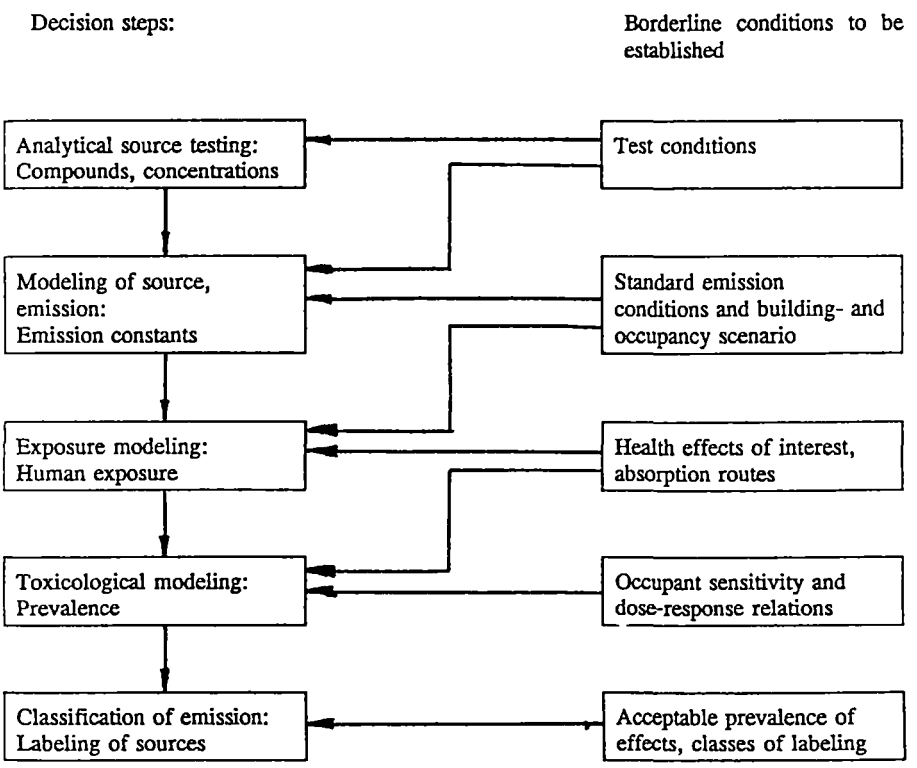
adequate information is available on compounds whose estimated contribution to disease is characterised.

5.6.2 EMISSION STANDARDS

The best way to achieve good indoor air quality and a reduction in VOCs is source control. According to Maroni *et al.* (1995), it is necessary to firstly define acceptable product emissions. This can be done by determining a tolerable indoor concentration level, and with a number of assumptions regarding the average conditions under which the product is used in practice, the desired emission rate of a compound can be calculated, and a guideline value established. Conditions for such tests have to be carefully undertaken following guidelines to obtain data of comparable quality (American Society for Testing Materials 1989). The complexity of formulating regulations for product emissions is illustrated by Molhave (1995) in figure 5.5, which outlines the principal decision steps required in the regulation of emissions from building materials.

FIGURE 5.5

Principle decision steps in regulation of emissions from building materials
(Source: Molhave 1995, p29)



Australia uses a combination of standards and guidelines for the control of various pollutants (Standards Australia 1998). To date, the only standard available for material emissions is for formaldehyde in building materials (AS1859 (1996) Part 1, Reconstituted woodbased panels: decorative overlayed wood panels, AS1859 (1997) Part 2 Reconstituted woodbased panels: particle board, AS1859 (1997) Part 3 Reconstituted woodbased panels: medium density fibre board). Similar control measures (standards/guidelines) would undoubtedly be available in other countries, but information is difficult to obtain.

5.7 Conclusion

Legislation would provide a firmer basis for ensuring compliance with indoor air quality protocols. These various approaches may include annual audits of buildings, an increased uniformity in occupational health and safety issues, and new processes for the implementation of standards throughout Australia and the world. Parallel to these activities could be such activities as public health campaigning.

Guidelines or standards for chemicals are developed on a pollutant by pollutant basis and do not consider low levels of contaminants or the interactions that may occur in synergistic multi contaminant environments. The TVOC concept addresses this issue and is useful as a measure of exposure by providing a basis for categorising VOC levels and their health effects. To use it as a indicator of potential health risk or as a provisional guideline still needs to be justified.

Overall, more exposure estimates on individuals should be assessed. This is especially the case for exposures caused by complex sources and noncarcinogenic substances. We are also yet to determine the precise relationships between pollutants and health effects, but where information exists, feasible exposure limits should be set in accordance with this.

The key to controlling indoor air pollution and volatile organic compound exposure is to prevent any future problems. To do this we need a harmonisation of efforts to ensure the protection of individuals. Legislation, standards and guidelines relevant to a range of indoor environments appear to be among the most appropriate means of achieving this.

CHAPTER SIX

VOLATILE ORGANIC COMPOUND INVESTIGATIONS - A REVIEW

6.1 Introduction

Volatile organic compound investigations in non-industrial buildings have developed following traditional occupational health and toxicological methodologies.

Repeated tests on VOCs in indoor air have established the presence of gas/vapour phase contaminants and their implications to physical well-being. Yet, the diverse range of analytical procedures and sampling techniques applied makes direct comparisons (on organic contaminants) between studies difficult.

A review of past and current research is necessary in order to examine methods commonly used in VOC analysis, and their subsequent results. An evaluation of available information places this Tasmanian study in the context of other VOC investigations seen throughout the world. Studies discussed rely predominantly on methodologies and experiments made outside Australia, as very little published material of Australian origin exists.

An overview of the literature on VOCs highlights three major areas of methodological interest. Exposure studies are the most widely used, and essentially demonstrate the potential health effects from exposures. Systematic building investigations are also applied extensively, and primarily ascertain whether VOC and TVOC levels are associated with SBS symptoms. The third most frequently cited studies are emission tests which determine the major VOC species (individually or in mixtures) in pollutant sources (e.g. materials and furnishings), and their emission properties.

6.2 Exposure Studies

6.2.1 PROGRESS TOWARDS VOC CHARACTERISATION

Exposure studies, based on inhalation experiments, were amongst the first methods used to demonstrate that buildings, especially new or renovated, can cause changes in perceived air quality, mucous membrane irritation and neurobehaviour.

A Danish group led by Molhave (Kjaegaard *et al.* 1991, Molhave *et al.* 1991, Molhave 1990, Molhave *et al.* 1993) conducted a number of human experiments to determine the potential effects of VOC mixtures in subjects who had previously complained of SBS. Other exposure investigations followed Molhave's initial study by various research teams, who confirmed results by identifying a relationship

between VOC exposure and mucous membrane irritation (Hundell *et al.* 1992, Koren *et al.* 1992, Otto *et al.* 1990, Otto *et al.* 1992).

Even though sensory irritation was evident, many were unable to replicate these earlier experiments in regard to effects on neurobehaviour (Otto *et al.* 1990, Otto *et al.* 1992). Another major criticism of the study has been the choice of test subjects which were thought to represent extreme cases and sensitive individuals. To eliminate this problem additional studies were designed to be representative of normal building populations (Hundell *et al.* 1992, Koren *et al.* 1992).

Overall, the results found in the first VOC exposure studies by Molhave and his team provided a theoretical and methodological framework for many of the subsequent follow up experiments and VOC testing procedures.

Current research is very similar in procedure to these initial studies, with the only difference being more specialised assessment of VOC species, their chemical transformations, and physiological effects. For example, Eide and Zahlsen (1996), conducted inhalation experiments in human subjects exclusively examining aromatic hydrocarbon mixtures at varying vapour levels. Similarly Knudsen *et al.* (1997) used a sensory panel to assess perceived air quality following an exposure to eight furnishing materials often found indoors. Some studies are even more distinct, like those undertaken by Kovacs *et al.* (1997) who analysed the relationship between chemicals used in cleaning, their odours and behavioural changes in 342 subjects.

Broader investigations are sometimes selected with the intent to confirm dose-response relationships. A study undertaken by Koren and Devlin (1992) is one such example, where human exposure studies were conducted to general VOC mixtures. Raaschounielsen *et al.* (1997) used a similar approach by evaluating overall personal exposures to benzene, toluene and xylene in Danish children in homes using diffusive samplers. In some situations a combination of techniques is applied to increase validity between correlations. This is illustrated in an investigation by Smedje *et al.* (1997), who employed a combination of VOC exposure measurements and questionnaires to 1410 subjects to compare health aspects.

Some elements in the Tasmanian research project discussed in this thesis are similar to a number of published exposure studies. For example, both the Raaschounielsen *et al.* (1997) study and this Tasmanian investigation examine VOC exposure using diffusive samplers. Similarly, comparisons can be made between the investigation by Smedje *et al.* (1997) and the Tasmanian study on office buildings, because of the combined use of quantitative and qualitative techniques.

6.2.2 ANALYSIS OF IRRITATION AND ODOUR EFFECTS

Molhave (1986) first identified that eye, nose and throat irritation increases during VOC exposure, suggesting that trigeminal systems are responsible for sensory effects. This hypothesis consequently initiated a new direction in VOC studies and the theory surrounding odour thresholds.

For example, Hundell *et al.* (1992) exposed 66 male subjects to clean air and VOC mixtures commonly found in new or recently renovated buildings. Findings revealed that intensity of perceived irritation was not related to odour intensity, but that subthreshold levels of compounds interact additively or hyperadditively to stimulate trigeminal nerve receptors. A follow up study by Hundell *et al.* (1993) confirmed these observed effects. Koren *et al.* (1992) examined exposures to known amounts of VOCs (25 mg/m^3 TVOC) in carefully controlled conditions representative of homes and office air. Using a nasal lavage to monitor neutrophil (PMN) influx into the nasal passages following exposure, statistically significant increases in PMNs were observed 4hr and 18hr after exposure.

To measure biological effects of VOCs, Walinder *et al.* (1997) undertook hygienic measurements of 27 school children applying an acoustic rhinometry technique. The degree of swelling of the nasal cavity was measured, and results concluded that raised levels of indoor air pollutants caused subclinical swelling of the nasal mucosa. According to Walinder *et al.* (1997), the practical application of acoustic rhinometry in field studies could be a more sensitive and objective measure of biological effects.

Olfactory models and more sensitive analytical methods for estimating irritation have been subsequently developed using the above mentioned techniques. Comettomuniz and Cain (1998) gauged evidence of human nasal irritation to VOCs by examining alternative psychophysical methods. Their study focused on a comparison of modalities and methods of measurement, by evaluating a common detection procedure versus a nasal laterisation procedure. Results revealed that the method for stimulus representation had little effect on measures of irritative or olfactory detection, therefore both procedures are suitable for normosmic and anosmic individuals. Similarly Hau and Connell (1998) developed a theoretical model for correlating odour thresholds of VOCs with their physicochemical properties. Results indicated that acetates, alcohols, ketones, and amines bind to a common receptor site located in the hydrophobic interior of the lipid bi-layer membrane of the olfactory cilia.

Contrastingly, this Tasmanian study does not expose building occupants to known concentrations of VOCs, but alternatively examines in the field if levels of VOCs are correlated to sensory irritation. Unlike the majority of controlled exposure studies, this Tasmanain investigation could possibly be a better indicator of what exposures are encountered in a building that is occupied, and the health

effects. Controlled exposure studies are useful in determining effects resulting from defined VOC concentrations (upper and lower extremes), but this is not representative of exposures encountered during the course of a day. For example, the VOCs measured in controlled exposure studies are assessed in isolation which do not take into consideration fluctuations in concentrations and interactions with other indoor air factors e.g. temperature and humidity. In contrast this "isolated assessment" can, in some instances, be perceived as an advantage because it eliminates interactive effects in a study.

6.2.3 BIOLOGICAL EVALUATION

Biological evaluation of contaminants in humans and rodents (Stadler & Kennedy 1996, Tap *et al.* 1996) have been utilised before in toxicology, but only recently applied in the assessment of VOCs.

Analysis of biological fluids is seen as a convenient and accurate way of quantifying exposure. This is illustrated by Hung *et al.* (1998), who used thermal desporption gas chromatography and mass spectrometry (GC/MS) to analyse unmetabolised benzene, toluene, ethylbenzene, and xylene levels in urine to assess VOC exposure. Unfortunately whether this technique is reliable and valid as a biological marker remains in question. The majority of VOCs have no positive marker for disease therefore these types of results convey no information on disease risk (Wilcosky 1993). The exception to this is the confirmed biomarkers present following PAH exposure (Jongeneelen 1997).

Researchers like Brooks and Riviere (1996) take a more direct analytical approach to assessing chemical exposure. Because skin remains an important route by which toxicity may occur, they implemented topical experiments on skin to phenolic compounds to evaluate systemic toxicity. Alternatively, researchers like Tafazoli *et al.* (1998) use other biological samples, in this case isolated human lymphocytes, to analyse the genotoxic and cytotoxic potential of VOCs.

Blood products are also useful in evaluating organic chemical exposure (Ashley *et al.* 1994, Chang *et al.* 1993, Dejongh *et al.* 1997, Dills *et al.* 1994, Kostrzewski 1993). Mannino *et al.* (1995) state that VOCs have been proven to be measurable in blood and the breathing zone of humans. Results highlighted that VOC levels found in organic vapour badges were highly correlated with blood levels of the same compounds. Therefore the feasibility of using whole blood as an indicator is one of the most sensitive methods designed to assess low levels (low parts per trillion) (Cardinali *et al.* 1995) of exposure.

Many authors use a combination of techniques for biological evaluation, but the more favored techniques are tenax traps to initally collect samples followed by infrared spectrometry, capillary gas chromatography, high performance liquid chromatography (HPLC) and ion-trap detection to determine concentrations

(Cardinali *et al.* 1995, Ojanpera *et al.* 1996, Schuberth 1996). Organic vapour badges are also used to gather ambient measurements, and are in some cases preferred because they are believed to be more sensitive than conventional charcoal tubes (Mannino *et al.* 1995). Personal monitors have the most promise for minimising the uncertainty about the degree of contact with a contaminant.

According to Angerer *et al.* (1997), analytical methods used thus far in estimating health risks to hydrocarbons lack accuracy by being nonspecific and are not comparable to one another. Only the use of very specific methods of instrumental analysis, above all gas chromatography/mass spectrometry (GC/MS) and high performance liquid chromatography (HPLC), can counteract any deficit. This is especially the case when evaluating DNA and protein adducts of PAHs.

6.3 Systematic Building Studies Examining VOCs

A number of investigations have been attempted to determine whether VOC levels are associated with SBS symptoms. The Danish town hall study (Skov *et al.* 1990, Skov & Valbjorn 1990) and Californian healthy building study (Fisk *et al.* 1993) failed to show relationships between VOC and retrospectively reported symptoms. This is in contrast to the findings of Hodgson *et al.* (1991), Hodgson *et al.* (1992), and Norback *et al.* (1990b) who observed positive correlations between SBS and the logarithmic value of TVOC concentration. According to Alarie (1981), the log-linear relationship between VOC levels and SBS symptoms is consistent with toxicological studies using mouse bioassays. Baird *et al.* (1987) extends on these studies even further by being able to distinguish between "sick" and "healthy" school buildings by means of chemical classification (presence or absence of some VOC types).

The use of field studies is increasing and now incorporates a wide variety of building types including schools, mobile homes, colleges, workplaces (especially industrial) (Valicenti & Wenger 1997), and private residences (Reiss *et al.* 1995). The office environment or non-industrial workplace on the other hand is an area that has been less adequately dealt with (Chuah 1997, Jaakkola 1998). Therefore this Tasmanian study contributes some much needed information on offices and the VOCs present in these non-industrial indoor environments. In addition, it complements the above mentioned systematic building investigations by assessing correlations between SBS and TVOC/VOC concentrations in occupied buildings.

According to Fanger *et al.* (1988), aside from variability in building types, there is also poor correlation problems with different sampling techniques. Molhave (1998) reinforces this view by stating that there needs to be a consensus for simplified procedures to identify and quantify compounds of relevance to indoor

air. This same problem emerges with the TVOC model where different definitions and different techniques yield different results. Therefore its usefulness as a predictor is in question (Molhave *et al.* 1997).

Regardless of varying methodologies many building investigators have proceeded in evaluating VOCs. As with exposure studies a combination of techniques have been adopted for systematic building investigations. For example, Molhave *et al.* (1996) modelled emission rates of material samples found in apartments by using laboratory measurements together with actual building measurements. Overall, the most frequently cited monitoring techniques for hydrocarbons are Tenax traps, granular activated carbon (Vanosdell *et al.* 1996), and passive monitors (Dingle *et al.* 1994). Subsequently organic compound concentrations are chemically analysed with GC/MS (Brown 1996, Chuah *et al.* 1997, Igielska *et al.* 1995).

Alternatively, Stewart *et al.* (1998) found job specific questionnaires successful for case control studies and to assess level of exposure. Drahonovská and Gajdos (1997) implemented a similar technique by combining questionnaires and measurements in dwellings and preschools. Wieslander *et al.* (1997) confirmed relations between asthma and emissions from newly painted surfaces by administering questionnaires coupled with measured formaldehyde concentrations. Wolkoff *et al.* (1998), on the other hand, combined lab emission testing and field studies to evaluate the use of cleaning agents and overall VOC levels.

The advantage in systematic building studies is that other extraneous factors can be included in the sampling procedure, which otherwise would not be evident in controlled laboratory conditions. For example, Farrow *et al.* (1997) used a longitudinal study to monitor VOC exposure based predominantly on observational techniques. Similarly, Gustafsson *et al.* (1996) related asthma and hypersensitivity to observable condensation rates found in certain types of homes. Quantification of these condensation rates could only be assessed in buildings that were occupied. This is also the case when comparing indoor/outdoor VOC concentrations (Drakou *et al.* 1995, Fishbein 1992).

6.4 Emission Tests

Chemical emission testing and modelling is the newest direction in VOC identification (Levin 1991, Wolkoff & Nielsen 1996). These techniques aim to identify and categorise building materials that are free of chemical emissions and unfavourable sensory properties (Maroni & Lundgren 1998). They also serve as a novel approach for point source measuring (Knaebel & Yeoman 1997). Countries like Germany, Denmark, Sweden, and Finland, alongside the European

Collaborative Action group, have already proposed a criteria on VOC emissions sensory testing coupled with toxicological evaluation.

The majority of emission tests centre around GC/MS to identify and quantify VOCs (Cooper *et al.* 1995, Darrall *et al.* 1998, Hodgson 1995, Seko & Onda 1997). This is illustrated in table 6.1 which reviews published total emission rates determined by GC/MS (using FID) (Maroni *et al.* 1995). Even more recently Lindinger *et al.* (1998) used a proton transfer reaction mass spectrometer (PTR-MS) to allow measurements of trace components with concentrations as low as a few parts per trillion (PPTV). This added sensitivity is of great advantage in quantifying low level organic pollutants outgassed by materials.

Even though GC/MS alone remains popular for ambient air tests, it is being superceeded by test chamber techniques that yield more accurate results for outgassing contaminants. Because samples are isolated in test chambers they eliminate interactive factors, and essentially reduce error. There are many uses for test chambers including simulation of workplaces (Meininghaus *et al.* 1998), and as a tool for validating models (Lennert *et al.* 1997). For example, Crump *et al.* (1997) measured VOCs in building materials for two years in test houses during construction and refurbishing. Samples collected were subsequently tested in test chambers to evaluate compounds outgassed. Many researchers use test chambers for evaluation of specific materials like furniture coatings and textile finishes (Guo *et al.* 1998, Martin *et al.* 1998, Muller & Schaeffer 1996, Reitzig *et al.* 1998, Salthammer 1997). Unfortunately, according to Leovic *et al.* (1996), no standard test method exists for evaluating emissions from office equipment and other furnishings. Some use it in combination with other techniques like Keller and Beckert (1994) who coupled material emission testing with ambient air sampling in a new building. It may also be successful in evaluating sorption, desorption and sinks of indoor materials (Vanderwal *et al.* 1998).

In contrast, Sparks *et al.* (1996) claim that emission test rates cited in publications are determined by chamber testing, which is then fitted to an empirical model. In their opinion, data resulting from this technique therefore does not provide the information necessary to scale it to buildings.

Emission tests have mainly been used on carpet samples due to its controversial nature as a toxic building material. Tepper *et al.* (1995) have shown with emission testing that the offgassing of carpets causes sensory and pulmonary irritation. An alternative view is presented by Dietert & Hedge (1996), who believe that VOC emissions are low enough, when reviewing the literature, that they should not significantly affect indoor air quality or pose as a significant risk to health.

TABLE 6.1

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Floor covering				
Carpet	floor and wall covering, textile	83	new	Molhave (1982)
Carpet	floor covering, synthetic fibres/PVC	120	new	Molhave (1982)
Carpet	carpet, UF backing (1 hr)	411	no seam	Davidson *et al.* (1991)
Carpet	carpet, UF backing (1 hr)	62	direct from manufacturer	Davidson *et al.* (1991)
Carpet	carpet, UF backing (1 hr)	98	aged	Davidson *et al.* (1991)
Carpet	carpet, UF backing (24 hr)	202	no seam	Davidson *et al.* (1991)
Carpet	carpet, UF backing (24 hr)	35	direct from manufacturer	Davidson *et al.* (1991)
Carpet	carpet, UF backing (24 hr)	26	direct from manufacturer	Davidson *et al.* (1991)
Carpet	carpet, UF backing (140 hr)	20	direct from manufacturer	Davidson *et al.* (1991)
Carpet	carpet, UF backing (140 hr)	111	no seam	Davidson *et al.* (1991)
Carpet	carpet, UF backing (140 hr)	6	direct from manufacturer	Davidson *et al.* (1991)
Carpet	felt carpet	80	new	Molhave (1982)
Carpet	latex backed carpet (336 hr) 4-PC	80		Tucker (1988b)
Carpet	carpet, SBR latex backed (144 hr)	45	direct from manufacturer	Black *et al.* (1991)
Carpet	carpet	36	<92 days	Wallace *et al.* (1987)
Carpet	felt carpet (synthetic fibres/ plastic backing)	11	new	Molhave (1982)
Carpet assembly	carpet, adhesive 3 on concrete (24 hr)	153000	single stick new product	Black *et al.* (1991)
Carpet assembly	carpet, adhesive 1, pad 1 on concrete (24 hr)	145000	double stick	Black *et al.* (1991)
Carpet assembly	carpet, adhesive 1 on concrete (24 hr)	98000	single stick new product	Black *et al.* (1991)
Carpet assembly	carpet, adhesive 2 on concrete (24 hr)	88300	single stick new product	Black *et al.* (1991)
Carpet assembly	carpet, adhesive 4 on concrete (24 hr)	783	single stick new product	Black *et al.* (1991)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Carpet cushion	carpet, pad 3 on concrete (24 hr)	775	no adhesive new materials	Black *et al.* (1991)
Carpet assembly	carpet, pad 1 on underlay (24 hr)	549	no adhesive new materials	Black *et al.* (1991)
Carpet assembly	carpet, pad 1 on concrete (24 hr)	136	no adhesive new materials	Black *et al.* (1991)
Carpet cushion	cushion P-3 (144 hr)	8110	new from factory	Black *et al.* (1991)
Carpet cushion	cushion P-3 (24 hr)	3360	new from factory	Black *et al.* (1991)
Carpet cushion	cushion P-2 (24 hr)	240	new from factory	Black *et al.* (1991)
Carpet cushion	cushion P-1 (24 hr)	123	new from factory	Black *et al.* (1991)
Carpet cushion	cushion P-1 (144 hr)	59	new from factory	Black *et al.* (1991)
Carpet cushion	cushion P-2 (144 hr)	12	new from factory	Black *et al.* (1991)
Vinyl	vinyl floor covering	22280		van der Wal *et al.* (1990)
PVC	Central european PVC	7034	1-3 yr, subject of complaint	Saarela & Sundell (1991)
PVC	floor cover, homogenous PVC	2300	new	Molhave (1982)
PVC	Finnish PVC covering	2192	0.5 yr, unused	Saarela & Sundell (1991)
PVC	Finnish PVC covering	1629	1 yr, unused	Saarela & Sundell (1991)
PVC	Finnish PVC covering	1443	<0.5 yr, unused	Saarela & Sundell (1991)
Rubber	floor covering, rubber	1400	new	Molhave (1982)
PVC	English PVC covering	1122	1 yr, unused	Saarela & Sundell (1991)
PVC	Finnish PVC covering	273	2-3 yr, subject of complaint	Saarela & Sundell (1991)
PVC	Swedish PVC covering	91	1-2 yr, subject of complaint	Saarela & Sundell (1991)
Vinyl	vinyl tile	45	age <98 days	Wallace *et al.* (1987)
Soft plastic	floor covering, soft plastic	590	new	Molhave (1982)
Linoleum	linoleum floor covering	220	new	Molhave (1982)
Linoleum	linoleum	64	30 yr subject of complaint	Saarela & Sundell (1991)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Linoleum	floor covering (linoleum)	22	new	Molhave (1982)
Wood	pine, industrial (1 mo)	682	1 yr surface coating	Saarela & Sundell (1991)
Wood	birch, industrial	272	1 yr surface coating	Saarela & Sundell (1991)
Wood	pine	264	1 yr UF lacquered	Saarela & Sundell (1991)
Wood	pine, untreated	216	new, in plastic wrap	Saarela & Sundell (1991)
Wood	birch, industrial	157	1 yr surface coating	Saarela & Sundell (1991)
Cork	cork	805	3 yr new material	Saarela & Sundell (1991)
Cork	cork	7	2 yr subject of complaint	Saarela & Sundell (1991)
Wet products				
Adhesive	wall and floor glue	271000	water based EVA	Molhave (1982)
Adhesive	adhesive, wall and floor (24 hr)	270000		Molhave (1982)
Adhesive	floor adhesive	220000		Tucker (1988b)
Adhesive	carpet adhesive (24 hr)	99000		Black *et al.* (1991)
Adhesive	carpet adhesive (24 hr)	9000		Black *et al.* (1991)
Adhesive	carpet adhesive (24 hr)	76600		Black *et al.* (1991)
Adhesive	carpet adhesive low VOC (24 hr)	698	low VOC formulation	Black *et al.* (1991)
Adhesive	carpet adhesive (144 hr)	17200		Black *et al.* (1991)
Adhesive	carpet adhesive (144 hr)	11900		Black *et al.* (1991)
Adhesive	carpet adhesive (144 hr)	3950		Black *et al.* (1991)
Adhesive	carpet adhesive low VOC (24 hr)	76	low VOC formulation	Black *et al.* (1991)
Adhesive	carpet adhesive (7 d)	234		Wallace *et al.* (1987)
Adhesive	cove adhesive (7 d)	5000	methanol based glue	Wallace *et al.* (1991)
Adhesive	primer/adhesive (7 d)	6.1	wall primer adhesive	Wallace *et al.* (1991)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Adhesive	floor adhesive (10-100hr)	<5000		Tucker (1988)
Adhesive	texture glue, PVA, water based (24 hr)	2100		Molhave (1982)
Seam sealant	carpet seam sealant	2960	seam sealant only	Davidson *et al.* (1991)
Seam sealant	carpet seam sealant	249	seam sealant only	Davidson *et al.* (1991)
Sealant	sealing agent plastic compounds (24 hr)	72000		Molhave (1982)
Sealant	sealing agent plastic compounds (24 hr)	26000		Molhave (1982)
Sealant	urethane sealant	0.13		Wallace *et al.* (1987)
Polish	spray polish for furniture	27100		Colombo *et al.* (1990)
Wax	floor wax (<10 hr)	80000		Tucker (1988)
Wax	floor wax (10-100 hr)	<5000		Tucker (1988)
Wax	floor wax paste (initial emission rate)	1880		Colombo *et al.* (1990)
Various building materials				
Fibre board	woodfibre board (12 mm)	120	new	Molhave *et al.* (1982)
Fibre board	fibre board, glass fibre polyester reinforce	17	new	Molhave *et al.* (1982)
Gypsum board	calcium silicate board	64	new	Molhave *et al.* (1982)
Gypsum board	plaster board	26	new	Molhave *et al.* (1982)
Gypsum board	plaster board (12 mm) paper coated	26	new	Molhave *et al.* (1982)
Gypsum board	gypsum board	26	age unknown	Molhave *et al.* (1982)
Gypsum board	water repellant mineral board	1.5	age unknown	Tucker (1988b)
Insulation	insulation foam polystyrene	1400	new	Molhave (1982)
Insulation	polystyrene foam A	260		Van der Wal *et al.* (1990)
Insulation	insulation foam, polystyrene	120	new	Molhave (1982)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Insulation	polystyrene foam b	30		Van der Wal *et al.* (1990)
Insulation	polystyrene foam insulation	22	retail new	Wallace *et al.* (1987)
Insulation	insulation batt (wool)	12	new	Molhave (1982)
Insulation	duct insulation	0.28	construction site	Wallace *et al.* (1987)
Caulk	silicone caulk (<10h)	13000		Tucker (1988)
Caulk	silicone caulk (10-100hr)	<2000		Tucker (1988)
Caulk	filler, PVA, glue cement	10200		Molhave (1982)
Caulk	filler, sand, cement (24hr)	730	water based	Molhave (1982)
Caulk	latex caulk (7d)	637	interior/ exterior	Wallace *et al.* (1987)
Caulk	sealing agent	340	new	Molhave (1982)
Caulk	tightening fillet (24hr)	160	neoprene	Molhave (1982)
Caulk	plasticised/PVC	56	new	Molhave (1982)
Caulk	heat expanding neoprene	17	new	Molhave (1982)
Caulk	tightening fillet	16	new	Molhave (1982)
Paint	acrylic latex	430		Molhave (1982)
Paint	latex high profile	249		Wallace *et al.* (1987)
Paint	vinyl flat white	3.2		Wallace *et al.* (1987)
Stain	wood stain (<10hr)	10000		Tucker (1988)
Stain	wood stain (1-100hr)	<100		Tucker (1988)
Varnish	polyurethane wood finish (<10hr)	9000		Tucker (1988)
Varnish	floor varnish (24hr)	4700		Molhave (1982)
Varnish	clear epoxy	1300		Molhave (1982)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Varnish	acid hardener (24hr)	830		Molhave (1982)
Varnish	polyurethane wood finish	<100		Tucker (1988)
Laminated borad	plastic	0.4	new	Molhave (1982)
Particleboard	particleboard (new)	2000		Tucker (1988)
Particleboard	particleboard (24hr)	952		Black *et al.* (1991)
Particleboard	particleboard (144hr)	837		Black *et al.* (1991)
Particleboard		200	2 years	Tucker (1988)
Particleboard		140	new	Molhave (1982)
Particleboard	urea formaldehyde glued	130	new	Molhave (1982)
Particleboard		120	new	Molhave (1982)
Particleboard		28	<98 days	Wallace *et al.* (1987)
Plywood	plywood b	1450		Van der Wal *et al.* (1990)
Plywood	plywood a	900		Van der Wal *et al.* (1990)
Plywood	plywood c	725		Van der Wal *et al.* (1990)
Plywood	plywood d	215		Van der Wal *et al.* (1990)
Sheathing		0.03		Wallace *et al.* (1987)
Panelling	plywood panelling HCHO	100	new	Tucker (1988)
Panelling	plywood panelling teak	44	new	Tucker (1988)
Wall covering	vinyl fibreglass	300	new	Molhave (1982)
Wall covering	wallpaper, PVC foam	230	new	Molhave (1982)
Wall covering	wall covering, PVC	100	new	Molhave (1982)
Wall covering		100		Tucker (1988)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Wall covering	vinyl coated	95	new	Van der *Wal et al.* (1990)
Wall covering	printed wallpaper	31	new	Molhave (1982)
Wall covering	vinyl coated	20		Van der Wal *et al.* (1990)
Wall covering	hessian	5.4	new	Molhave (1982)
Miscellany				
Cement block		0.54		Wallace *et al.* (1987)
Cement flag		73		Molhave (1982)
Cable	small diameter telephone	60		Wallace *et al.* (1987)
Cable	large diameter telephone	38	standard	Wallace *et al.* (1987)
Pipe	PVC pipe	0.53		Wallace *et al.* (1987)
Vapour barrier	tar pipe	6.3		Wallace *et al.* (1987)
Subfloor	concrete subflooring	<5		Black *et al.* (1991)
Trim	black rubber moulding	103		Black *et al.* (1991)
Cove base	vinyl cove moulding	46	<124 days	Wallace *et al.* (1987)
Cove base	vinyl edge moulding	30	<98 days	Wallace *et al.* (1987)
Textile	floor and wall covering	1600		Molhave (1982)
Cleanser	spray cleanser for carpets	50400	ınital emission	Colombo *et al.* (1990)
Cleanser	liquid cleanser/disinfectant	34900		Colombo *et al.* (1990)
Cleanser	liquid floor detergent	2200		Colombo *et al.* (1990)
Pesticide	moth cake (p-DCB) @23C	14000000		Tucker (1988)
Dry-cleaning	dry cleaned clothes (0-1 day)	100		Tucker (1988)
Dry-cleaning	dry cleaned clothes (1-2 days)	50		Tucker (1988)

TABLE 6.1 Continued

Emission rates for various building materials
(Source: Maroni *et al.* 1995, p39-48)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2h$)	Acquisition age	Ref
Office chair	high back with arms	1060	1 hr	Strobridge & Black (1991)
Office chair	high back with arms	100	981 hr	Strobridge & Black (1991)
Office furniture	tackable acoustical partitions	158	1 hr	Strobridge & Black (1991)
Office furniture	tackable acoustical partitions	74	2.5 hr	Strobridge & Black (1991)
Office furniture	tackable acoustical partitions	158	48 hr	Strobridge & Black (1991)
Office furniture	tackable acoustical partitions	6	581 hr	Strobridge & Black (1991)
Workstation		3200	1 hr	Strobridge & Black (1991)
Workstation		10	912 hr	Strobridge & Black (1991)
Workstation	HCHO	1470	1 hr	Strobridge & Black (1991)
Workstation	HCHO	830	336 hr	Strobridge & Black (1991)

6.5 Conclusion

Both the early small scale studies and current research combined has progressed towards the determination of the multitude of different VOCs found in indoor air. Despite the somewhat different protocols and methods there appears to be a general consensus of opinion on the types of VOCs and their typical concentrations. Although, at present there is inadequate scientific basis on which to base indepth VOC evaluation, particularly in regard to standardised methods of quantifying the emissions and diagnosing health effects.

CHAPTER SEVEN

METHODOLOGY

7.1 Introduction

Overall there is a paucity of literature examining VOC contamination and sick building syndrome in Australian office buildings (Kemp & Dingle 1994, Rowe & Wilke 1994, Williams 1992). This is especially the case for the state of Tasmania, where non-industrial indoor environments have received little research attention.

Tasmania has been greatly neglected from an indoor air quality perspective for a variety of reasons including limited resources and legislative weaknesses. As a result the indoor air quality (IAQ) status of Tasmanian office environments has only been examined once (Mesaros 1995, Mesaros 1996). Initial consultation with building owners and managers indicated that there was ongoing occupant discomfort and dissatisfaction with the internal environment. Volatile organic compounds were thought of as a possible antecedent agent to SBS, given the nature of the symptoms experienced by individuals.

To further investigate the aetiology of Hobart's high sick building syndrome levels a quantitative and qualitative study was undertaken to examine background levels of VOCs in standard office buildings.

7.2 Building Characteristics, Study Population and Site Selection

Sampled buildings were located in the Hobart central business district. Eleven offices were chosen from a list of buildings identified as having a very high rate of employees suffering from sick building syndrome symptoms from an earlier Hobart study (Mesaros 1995). It was necessary to use buildings known to have complaints of SBS to fulfil the hypotheses and objectives of this thesis. Buildings were selected using a systematic sampling technique where every fifth building was chosen from a list of 55 buildings. A control building (with no evidence of SBS) was also selected, using the same technique, to provide a comparison to the eleven offices. The buildings (ranging in ages of between 2-27 years) represented a broad cross section of office accommodation ranging from modern city high rise to one storey buildings, as illustrated in plate 3. The sample size was made as large as practically possible to reduce the sampling error associated with selecting a sample from a larger population. To eliminate recording, editing and calculating errors all data was processed three times.

The study population consisted of 206 office workers, 104 males and 102 females. Individuals were selected on the basis of their proximity to the sampling device. Quantitative and qualitative analysis was undertaken simultaneously in

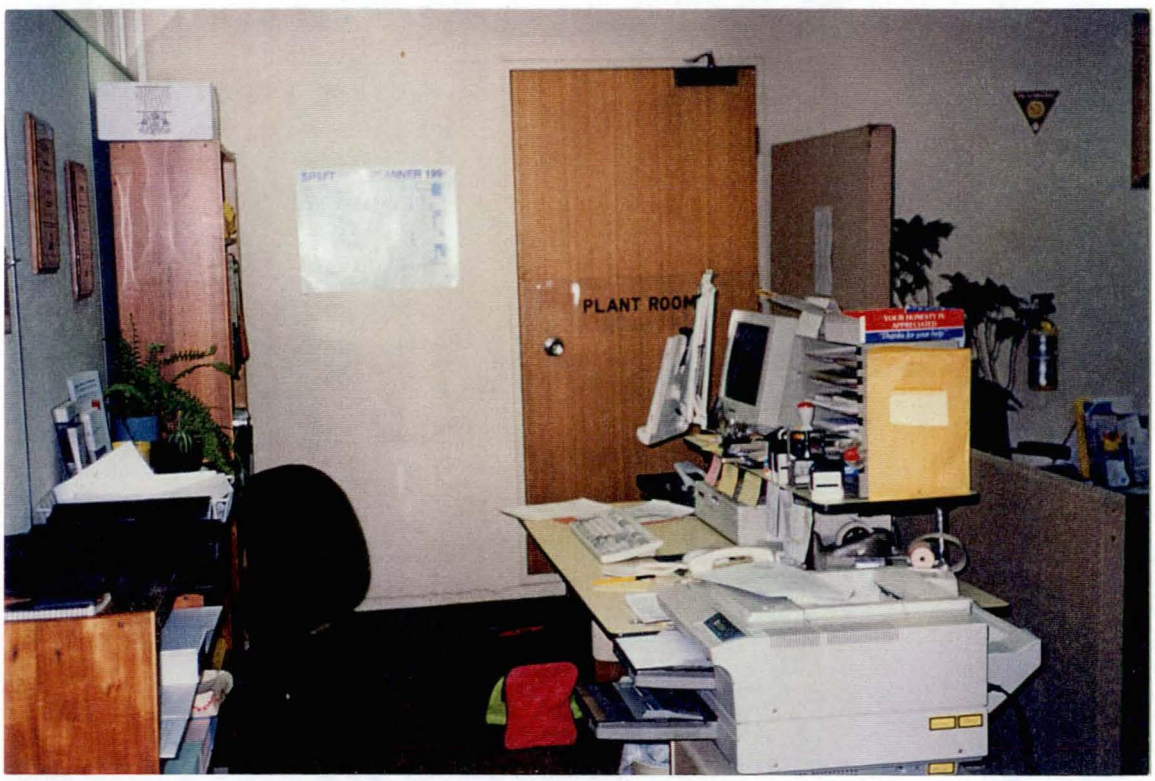
each building for 5 consecutive weeks in February/March (summer months) and 5 consecutive weeks in July/August (winter months).

All establishments had HVAC systems in operation during data collection, and no major disruptions to the offices were noted (refurbishment procedures, painting, re-carpeting) at the time of sampling.

In order to fulfill the objectives of this study, techniques, measuring devices and protocols employed are based on recognised standardised methods commonly used in occupational health and safety investigations. Where a standardised method has not been available, as is the case for many indoor air variables, procedures cited in the relevant literature were followed.

PLATE 3

Photograph of a typical multistorey office building in Hobart's central business district
(Location: 85 Macquarie St, Hobart 1998)



7.3 Quantitative Analysis

7.3.1 GAS DETECTION TECHNIQUES

The volatile organic compounds were sampled passively, using activated charcoal tubes, as illustrated in figure 7.1 and table 7.1 (Orsa 5, by Dräger) (Dräger 1994).

FIGURE 7.1

Illustration of the Orsa 5 sampling device
(Source: Dräger 1994, p51)

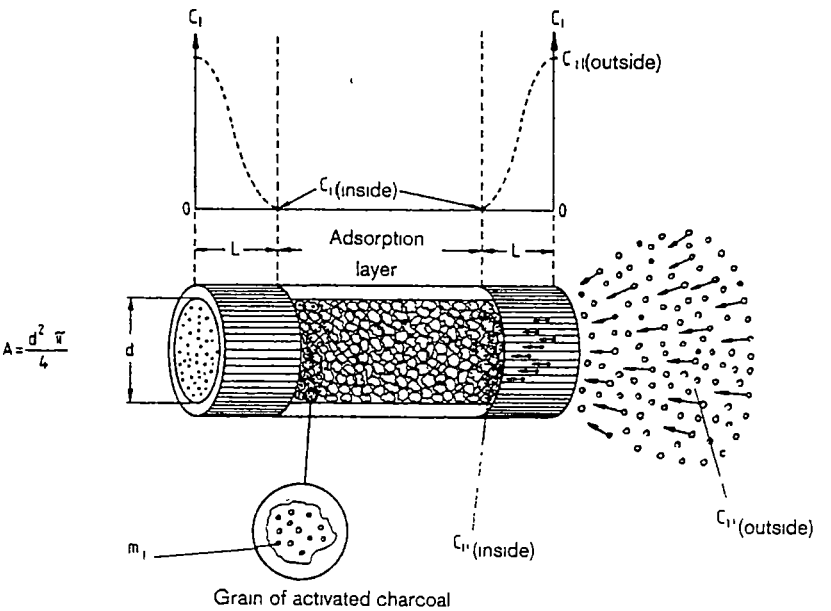


TABLE 7.1

Technical information regarding the Orsa 5 sampling device
(Source: Dräger 1994, p51)

Sampling medium -	Approx 400 mg coconut shell charcoal charcoal particle size 0.4 - 0.8 mm
Diffusion distance -	0.5 cm
Temperature -	0 - 40°C
Diffusion barrier -	Cellulose acetate
Time constant -	Approx 2 s
Diffusion rate -	1 - 4 µg/(ppm × h)
Atmospheric pressure -	Less than 1050 hPa
Humidity -	5 - 80% rel. humidity at 20°C

An important prerequisite in determining the concentration of any gaseous pollutant is the quantification of the concentration with a suitable gas measuring device. Since the diffusion tubes used do not require a pump, they are particularly effective for sanitary measurements in the non-industrial workplace (Dräger 1994). The range of application for these passive samplers is ideal for solvent analysis, as opposed to direct reading or colourimetric systems which cannot distinguish between types due to cross sensitivities.

The diffusion samplers are generally designed to sample over a long period of time for the determination of average concentrations. This is usually 1 to 8 hours, but in office and air sampling, monitoring can be continued for over 168 hours. The sampling time chosen for this study was seven days (168 hr) over a period of ten weeks for the following reasons:

1. The sensitivity of the method used for the quantitative analysis only allowed one weeks exposure. A minimum of seven days sampling was required for the detection of low substance concentrations present, but any longer than a week would have made it difficult to quantify some of the organic compounds present (due to evaporation off the charcoal) in the sampling device, therefore sampling never exceeded 168 hr;
2. Seven days was chosen as an appropriate time frame to assess average concentrations. It would have been preferable to include sampling of peak exposure (1-8hr) as well because when the concentration of a toxic substance varies rapidly with time, too long a sampling period will not measure the true concentration at the time of the exposure (Hang *et al.* 1992). Measurements of longer than seven days e.g. 1 month over a twelve month period would have highlighted a more realistic representation of average exposures but it was not possible using the quantitative method employed in this study;
3. One week was a sufficent time to make it convenient for employees to recall their symptoms easily; and
4. The costs involved in sampling everyday for ten weeks were beyond the budget of this study, therefore sampling once a week for ten weeks was seen as a suitable compromise.

Buildings were assigned a system of numbers for identification purposes. Numbers B1 1/5 - B11 1/5 represented buildings one to eleven during sampling weeks 1-5, and numbers B1 6/10 - B11 6/10 defined buildings one to eleven during sampling weeks 6-10. For both sampling weeks 1-5 and 6-10, the control building was identified by the letters "BC".

Samplers were sited in office buildings under normal working conditions and exposed continuously over seven days (168 hr), but workers were only present for

about 36-40 hr. During each of the sampled weeks, charcoal sampling tubes were placed in their respective locations between 10.30am-11.30am on Monday mornings and taken down seven days later at precisely the same time. Sampling devices were suspended from the ceiling with a length of string (approximately 1.5 metres from the ground), and were located relative to the people included in the questionnaire survey. Instead of capping the devices at the end of each working day, and uncapping them at the start of the day, samplers were left suspended in the workplace air for the full seven days. This method was selected because of the difficulty in gaining access to buildings after work hours, especially weekends.

Overall, steps were taken in all buildings, particularly multi-storey buildings, to avoid conditions that may adversely affect the performance of the sampling device, therefore samplers were sited in specific areas for the following reasons:

1. The sampling devices (particularly in multi-storey buildings) were located on a floor of the building which represented a "typical" office layout (open plan design with some partitioned areas);
2. The floors of the building where occupants spent most of their time were chosen as suitable sampling sites over floors that were unoccupied. For example, floors that were sparcely occupied or occupied only part of the time e.g. reception and conference areas, were not considered suitable because of the differing HVAC operations in these areas and low occupant densities;
3. To avoid extreme temperature variations neither the very core (HVAC compensates for internal heat gains, therefore the interior "core" spaces generally require year round cooling), nor the extreme periphery (HVAC compensates for indoor/outdoor temperature differences because of the exterior walls and ceilings) of the office floor was used to locate samplers. Instead, devices were sited away from these two spaces; and
4. It has been documented that the lower floor of a building is often affected by greater fluctuations in temperature and contaminant sources from outside (Maroni *et al.* 1995). Similarly, top floors are influenced by shifts in temperature because exterior walls or ceilings require additional heating or cooling as HVAC system compensates for various factors such as indoor/outdoor temperature differences. Therefore the ground and very top floors were avoided as sampling sites.

Once suspended, contaminant molecules from the ambient air were adsorbed by the coconut shell charcoal. Equations based on Fick's law of diffusion were then used for the calculation of the absorbed substance mass (Dräger 1994), as seen in figure 7.2.

Upon completion of sampling (168hr), diffusion tubes were capped, placed in glass storage jars, and taken to the laboratory immediately for analysis by staff in the Central Science Laboratory (CSL) at the University of Tasmania.

FIGURE 7.2

Calculation of concentration using passive samplers based on
Fick's law of diffusion
(Source: Dräger 1994, p51)

$$\Delta C_i = \frac{m_i \cdot L}{D_i \cdot t \cdot A} \left[mg/m^3 \right]$$

In this context:

m_i	The substance mass
t	Time
A	Sectional area of the sampler in which the substance diffuses vertically to the concentration grade
Δc_i	The concentration difference along the diffusion course, which is basically equivalent to the ambient concentration
L	diffusion course
D_i	The diffusion coefficient which is substance specific

7.3.2 ANALYSIS AND EVALUATION

Sampling tubes were analysed by solvent extraction, gas chromatography/ mass spectrometry using a Hewlett-Packard 5890 gas chromatograph and a Hewlett-Packard model mass selective detector instrument. The advantages to this technique are specificity for unequivocal identification, sensitivity and versatility for the separation for large numbers of compounds like hydrocarbons (Angerer *et al.* 1997, Hau & Connell 1998, Seko & Onda 1997). It has been demonstrated that gas chromatography/mass spectrometry (GC/MS) values are approximately 20% higher on average than other gas measuring techniques such as the flame ionisation method (FID), the photoacoustic infrared method (IR) and gas monitor method. Overall, evaluations of both GC/MS and FID procedures show similar precision (Hodgson 1995).

Analytical procedures followed were in accordance with the NIOSH and OSHA analytical methods, including NIOSH 1501 aromatic hydrocarbons (i.e. benzene, xylene, ethylbenzene and toluene), NIOSH 1500 (i.e. octane and hexane), NIOSH 1300 Ketones 1 (i.e. acetone), NIOSH 1003 (i.e. 1,1,1-trichloroethane), NIOSH 1400 Alcohols 1 (i.e. ethanol) (Dräger 1994, NIOSH 1994).

The sampling devices were removed from their holders, one of the porous damping layers from each of the tubes was removed by forceps, and the granular content trasnferred quantitatively to a sample bottle. After the bottles were closed with a cap, samples were desorbed using 3 mL carbon disulfide (CS_2) (added by means of a syringe), and a internal fixed standard, butyl butyrate $C_8H_{16}O_2$ ($CH_3CH_2CH_2COO(CH_2)_3CH_3$), added after extraction, to compensate for evaporation and response of CS_2. At the same time an unused sampling device from the same production batch was handled according to the same laboratory method so that any blind load value could be detected. Exothermic reactions in the charcoal tubes during the infiltration of samples with carbon disulphide, caused the aromatic hydrocarbon fractions to evaporate. This source of error has not been quantified. Advice from laboratory staff familiar with this technique (Dr Noel Davies, Laboratory Manager of GC/MS at CSL) suggest the error would be very small, and is common with this method.

Initial recovery experiments were undertaken to find response factors for standards before quantification of various compounds. Charcoal tubes were spiked with known concentrations of VOCs. These were then extracted and injected into the GC/MS. Standard solutions were injected directly into the GC/MS, and the ratio between the two results (spiked and pure standards) were compared to determine response factors. Calibrations were prepared using defined concentrations of 25 VOCs (appendix 3) in a blank sampling device, these were then compared to reagent standards in 3 mL of carbon disulfide to define the desorption efficiency. 1 µL of the solution was injected into the GC/MS by split injection into the column (HP-1 25 m x 0.32 mm, 7psi, film thickness 0.52 microns).

VOC concentrations were then determined by selected ion monitoring. Identification and quantification proved difficult because of dilute concentrations, with many levels on the limits of detection.

Results demonstrated a recovery of 1 for all the VOCs except acetone (.63) and Isobutane (.22). After GC/MS analysis, calculation of the average concentration of the measured substances was undertaken by further mathematical operations.

The concentration was calculated on the basis of the analysed mass of subtance m_1. Calculation of concentration (without pressure and temperature correction) (Dräger 1994) are as follows:

$$C_i = \frac{m_i \times K_{orsa\ 5}}{DA \times Di \times t}$$

C_i	Concentration of the contaminant measured ng/cm^3, corresponds to mg/m^3
Korsa 5	Devise constant of sampler = 0.8 cm $^{-1}$
m_i	Mass of the contaminant as determined in ng by gas chromatography, corresponds to 10 $^{-9}$ g
DA	Desorption efficiency of the measured substance (less than or equal to one)
Di	Diffusion coefficient specific to the substance in cm^2/s
t	Sampling time in seconds

The contaminant concentration was converted from mg/m^3 to mL/m^3 (ppm) (Dräger 1994) as follows:

$$C_i = [ppm] = C_i[mg/m^3] \times \frac{24.1}{\text{Molecular weight}}$$

7.3.3 TEMPERATURE AND HUMIDITY

Temperature and humidity values were analysed using a Visala direct reading monitor, and were taken where the samplers were suspended once a day at 12pm, when the buildings were occupied. Temperature and humidity readings were not able to be taken during the weekends because of difficulties in gaining access to buildings. Therefore, the measured temperature was not representative of the average 24hr building temperature. Multiple readings were taken at each VOC sampling point and converted to average values. Information and data on outdoor temperature and humidity (minimum, maximum and mean), were obtained from the Bureau of Meteorology (climate and consultancy section), on daily meteorological observations in Hobart (Ellerslie Road) (Bureau of Meteorology 1997).

It is also known that temperature, pressure changes and humidity affect the functioning of the activated charcoal in the sampling device (Dräger 1994). The temperature influence on the device is approximately 0.5%/K in the range of 0°C to 40°C, whereas the deviation due to pressure is approximately 0.1%/mbar in the

range from 980-1050 mbar. An equation was not used to correct the temperature and pressure effects on diffusion coefficients because atmospheric pressure was not measured during the sampling procedure. Therefore the diffusion coefficients used refer to organic compounds in air at 25°C and 1013 mbar (Dräger 1994).

To avoid effects of humidity on the absorption capacity of the activated charcoal, samplers were located at suitable sampling sites (i.e. not near dripping water), and were used within the acceptable humidity ranges (5% to 80% relative humidity at 20°C) however, the effect of humidity also depends on the substance to be measured. For example, a measurable effect of humidity on toluene measurements is not detected in a range of 5% to 95% relative humidity (Dräger 1994). Because humidity was in the acceptable ranges in this study no corrections were applied.

7.4 Qualitative Analysis

7.4.1 METHODS USED FOR SYMPTOM INQUIRY

The use of questionnaires is an integral part of indoor air and epidemiological investigations. Many studies (Borbeau *et al.* 1997, Dales *et al.* 1997, Eriksson *et al.* 1996, Menzies *et al.* 1998, Mikatavage *et al.* 1995, Norback 1995, Raw *et al.* 1996, Smedje *et al.* 1997) refer to the use of questionnaires in building investigations. In general they have been administered to rationally evaluate whether occupants are contented with a building, are exhibiting a high or low prevalence of symptoms, and to identify troublesome areas for further investigation.

However information resulting from questionnaires may be regarded as insufficient to assess the potential level of exposure accurately due to recall difficulties and the variablility of exposures leading to erroneous results (Stewart *et al.* 1998). In addition, factors such as pre-existing conditions, subjectivity, measurement scales, and psycholocial influences affect the quality assurance of questionnaire based investigations (Chang *et al.* 1993).

For this study a building information questionnaire, based on a combination of the Danish Research Building Institute questionnaire and the World Health Organisation building investigation procedures, was designed (appendix 4) (SBI 1990, WHO 1986, WHO 1987, WHO 1989). These questionnaires are most commonly used overseas and are designed to assure standardised data collection. The questionnaire addressed such building issues as floors, walls, ceiling, heating, lighting, room density, furniture, smoking, cleaning regimes, air conditioning and other environmental determinants. The questionnaire's format is general, but incorporates the major variables and areas of concern used when assessing indoor

air quality. The intention was to select only the most important building variables. The building questionnaire was completed for each building at the end of the sampling of weeks 1-5 and 6-10.

Self administered questionnaires were used for this study to identify symptoms in employees (appendix 5). As with the building information, a questionnaire targeting symptomatology was designed, based once again on the Danish Research Building Institute's questionnaire. It addressed the most common sick building syndrome symptoms, past disease history and general work environment determinants e.g. psychosocial factors. A sample questionnaire was given to a pilot group of subjects to determine the suitability of the questions and to evaluate the general layout of the questionnaire. Symptom questionnaires were distributed prior to the 5 week sampling period, to all building occupants (206 in total) who were near to the suspended sampling device. Each individual received five questionnaires and was instructed to record symptoms which were representative of "average" or "typical" conditions for the preceding week rather than conditions for a "single day". Respondents were asked to fill in the questionnaire once a week (Monday mornings), paying particular attention to what symptoms they were experiencing. Because such a large population group was sampled, five questionnaires (one for each week) were distributed at the start of the sample period.

In exposure assessments the best location for a sampler is next to or near the person, or group, whose exposure has to be determined. Therefore a selected sample of employees were used because of practical constraints, such as the impracticality of measuring every occupant in the building and the fixed locations of the sampling device. In addition, the decision of which employees were to be included had to be taken in light of the objective of this study which was to identify a relationship between SBS and the physical-chemical variables, so employees on other floors of the building or in locations not within the vicinity of the sampling device could not be included. The use of non-randomly chosen subjects is common in many systematic building investigations (Maroni *et al.* 1995, Molina *et al.* 1989, WHO 1986), and because of this some sampling bias would have undoubtedly affected the results of the questionnaire survey.

Individuals were asked to complete the questionnaire by recalling and detailing symptoms experienced during the sampling week. According to Maroni *et al.* (1995) in several circumstances recalling an event or behaviour of the past can be difficult in systematic building investigations, these include:

1. If the decision was made almost mindlessly in the fist place;
2. If the event was so trivial that people have hardly given it a second thought since;
3. If questions refer to events that happened long ago; and

4. If recall is required of several events.

To ensure the validity, reliability and quality assurance of the results from the questionnaire survey using a recalling of the past technique, the following points, as outlined by Maroni *et al.* (1995), were taken into consideration prior to sampling, these were:

1. The assessment of the health problems of a building was based on the whole working group or random selection of workers located at or near the sampling area and not on volunteers;
2. The questionnaires were simple and interlinked, complex or multiple part questions were avoided;
3. The same questionnaire was applied to all sampled individuals;
4. The questionnaire was based on other published questionnaires with the addition of a few new questions significant to the quantitative part of the study e.g. the introduction of symptoms such as liver pain;
5. A control building and control group were included in the study design;
6. The questionnaire included a question specifically addressing the building or work relatedness of symptoms; and
7. A standard procedure for the questionnaire responses was introduced, and a standardisation of questions, particularly the core questions. As with all building investigations, procedures for quality assurance was used throughout the sampling regarding design, data collection and data treatment.

The focus of the questions was to gauge averaging or typical conditions rather than variations over a single day. The completed questionnaire was then collected after the 5 sampling weeks.

7.5 Statistical Analysis

Statistical analyses have been conducted using the SPSS® program (SPSS Inc. Software 1990). Because the data was not normally distributed, non-parametric tests were predominantly used in the analysis. In some instances it was possible to transform the data to a natural logarithmic transformation, essentially creating a normal distribution. When this process was successful, parametric techniques were applied for statistical evaluation.

Prior to any statistical analysis the data was screened and transformed to comply with underlying generic assumptions that were relevant to the particular

statistical test being undertaken e.g. scale of measurement, normality, linearity, homoscedasticity, independence of observations etc. This included the rectification of errors in data, data entry recoding, collapsing continuous variables into categorical variables (e.g. median split), recoding negatively worded scale items and bringing outlying cases into the distribution.

7.5.1 PARAMETRIC TECHNIQUES

A Pearson correlation coefficient was used to describe the relationship between variables in both the qualitative and quantitative data (symptom and building questionnaire). A simple bivariate correlation was used instead of a partial correlation, as it is the preferred method of measuring a linear relationship between two data sets. The Pearson correlation coefficient is illustrated in figure 7.3.

FIGURE 7.3

The Pearson correlation coefficient (r)
(Source: SPSS Inc Software 1990)

$$r = \frac{\sum_{i=1}^{N} (X_i - \bar{X})(Y_i - \bar{Y})}{(N-1) S_x S_y}$$

N	Number of cases
S_x	Standard deviation of variable x
S_y	Standard deviation of variable y
$X_i - X$	The deviation of a X score from the population mean of all the X scores
$Y_i - Y$	The deviation of a Y score from the population mean of all the Y scores

FIGURE 7.4

The Pearson Chi-square statistic
(Source: SPSS Inc Software 1990)

$$X^2 = \sum_i \sum_j \frac{(O_{ij} - E_{ij})^2}{E_{ij}}$$

O_{ij}	Actual frequency in the i-th row, j-th column
E_{ij}	Expected frequency in the i-th row, j-th column
r	number of rows
c	number of columns

The Chi-square test for relatedness/independence was also applied using the Pearson's Chi-square statistic, this was used to determine if hypothesised results were verified the experiment. The Pearson's Chi-square statistic is illustrated is illustrated in figure 7.4.

7.5.2 NON-PARAMETRIC TECHNIQUES

Five different non-parametric techniques were used for data analysis, as there were serious violations from the normal distribution in the raw data. These tests tend to be less powerful than their parametric counterparts, but are appropriate for data measured on scales which are not interval or ratio. They are useful because they do not rely on parameter estimation or assumptions about parameters or the shape of the distribution.

The Mann-Whitney test (Wilcoxon rank sum) was used to highlight sex differences in symptom reporting. The Mann-Whitney test tests hypotheses that two independent samples come from populations having the same distribution. That is, this test is equivalent to the independent groups t-test.

Wilcoxon Signed-Rank test was also applied to both the qualitative and quantitative data, and is commonly refered to as the Wilcoxon T test. It is used in the same situations as one would use the Repeated Measures or Paired t-test. The Wilcoxon test was particularly useful in determining seasonal variations in symptom reporting and TVOC concentrations.

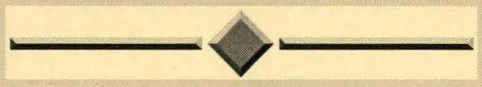
The Kruskal-Wallis test is equivalent to the one-way between groups analysis of variance (ANOVA) and thus allows the examination of possible differences

between two or more groups. This test was particularly successful in determining if factors such as year of employment affected symptom reporting in subjects.

The Friedman test was applied to the quantitative and qualitative data, particularly symptoms, VOCs and TVOCs, to ascertain if differences occured across observed results. This test is normally used to compare two or more related samples and is equivalent to the repeated measures or within-subjects ANOVA.

Spearman's rank-order correlation (Speraman's rho) was used extensively. This test is the non-parametric alternative to the bivariate correlation (Pearson's r). Most correlations between variables, especially TVOC and symptoms were analysed using this technique. Spearman's rank-order correlation was also applied to TVOC and building variables to see whether correlations were evident, but in addition to this tecnhique the Chi-square test for Goodness of Fit was applied to the results of the building questionnaire to determine if significant differences exist across response categories.

CHAPTER EIGHT



RESULTS PART ONE - FINDINGS of the QUANTITATIVE ANALYSIS

8.1 Introduction

This chapter presents the results of the quantitative investigation of this Tasmanian study following the methodology discussed in chapter seven.

A total of 112 samples were collected during the ten sampling weeks. Between 12-15 compounds were unequivocally identified and quantified in this study. They included several aliphatic hydrocarbons (alkanes), unsaturated cyclic hydrocarbons (aromatics), halogenated hydrocarbons, and oxygenated hydrocarbons (alcohols and ketones).

Hydrocarbons looked for but not detected in the samples included trichloroethylene, perchlorethylene, 1,2-dichloroethane, chloroform, styrene, hydroxybenzene, butylhydroxytoluene, *n*-hexane, naphthalene, glycol-n-butyl ether, ethylene glycol mono butyl ether, ethylene glycol mono methyl ether, 1,1,2,2-tetrachloroethane, and *n*-propanol.

The methodology used for the quantitative analysis in this study permitted only mean concentrations of compounds to be calculated, therefore individual and total organic compound levels discussed in this chapter represent average concentrations over the 7 sampling days.

8.2 Individual Analysis of Volatile Organic Compounds

8.2.1 COMPOUNDS IDENTIFIED AND THEIR CONCENTRATIONS

Compounds identified from the GC/MS were benzene, ethylbenzene, 1,2,4-trimethylbenzene, toluene, *m*-, *p*-, *o*- xylenes, *n*-decane, *n*-nonane, *n*-octane, limonene, α-pinene, 1,1,1-trichloroethane, ethanol, *n*-butane, isobutane, and acetone (for a detailed list refer to appendix 6 and appendix 7). Other trimethylbenzenes were also present but not quantified individually.

The control variable (reagent blank) consistently had traces of acetone, benzene and toluene. It was postulated that the carbon disulphide was contaminated by an unknown source even though the solution was a GC quality eluent. Further analysis of the carbon disulphide solution confirmed this finding. Therefore acetone (0.360 mg/m^3), benzene (0.679 mg/m^3), and toluene (0.381 mg/m^3) values from the control variable were subtracted from the other samples before calculation into mg/m^3.

As seen in table 8.1, the compounds with the highest detected levels were ethanol, ethylbenzene and *n*-butane for the whole 10 weeks of sampling. Compounds with the lowest levels present were α-pinene, limonene and *n*-octane.

To examine this further, the Friedman Two-Way ANOVA test was applied to see whether there was a statistically significant difference across detectable concentrations each week, during the ten week sampling period. General results indicated that VOC concentrations differed considerably (<.05). Ethanol levels were significantly higher in concentrations when compared to all other compounds (mean rank=13.75). This was followed by *n*-butane (mean rank=11.15), 1,1,1-trichloroethane (mean rank=11.15), and 1,2,4-trimethylbenzene (mean rank=10.15). Compounds with the lowest detected concentrations were *n*-octane (mean rank=3.25), benzene (mean rank=4.10) and limonene (mean rank=4.75).

TABLE 8.1

Identified compounds, their arithmetic means, ranges and percentile values (BLD represents concentrations "below the detection limit")

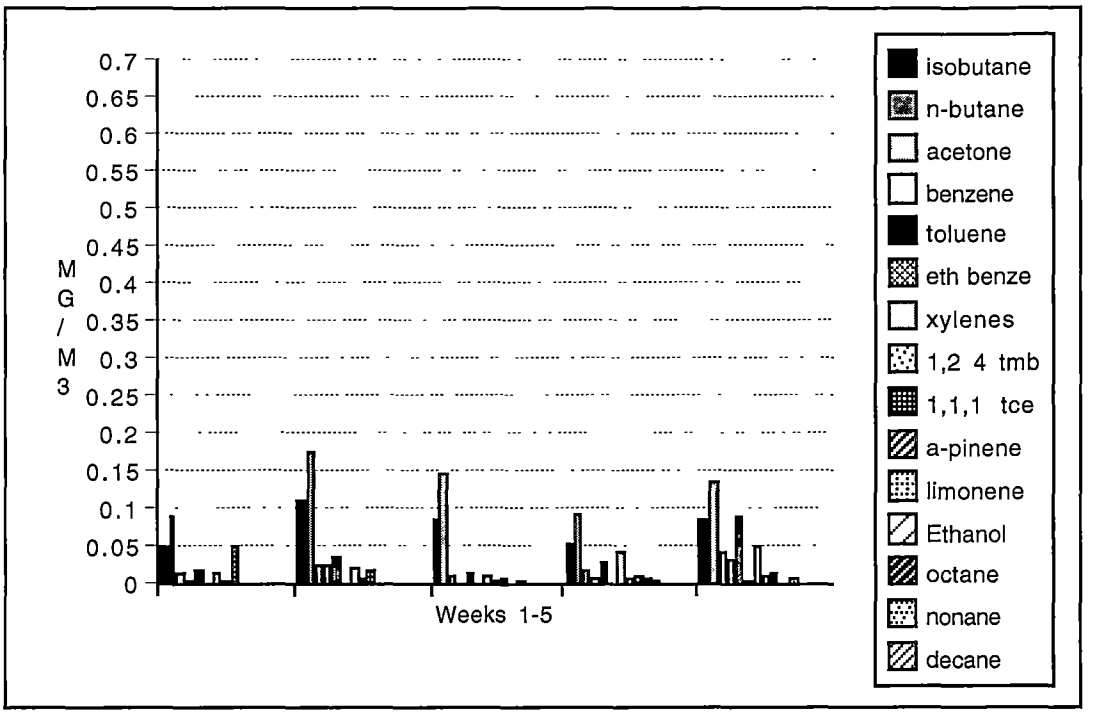
Compound	Arithmetic mean mg/m^3	Range mg/m^3	80 th Percentile	95 th Percentile
Isobutane	.031	BLD - .400	.042	.083
n-Butane	.045	BLD - .539	.057	.122
Acetone	.018	BLD - .059	.028	.049
Benzene	.010	BLD - .155	.013	.020
Toluene	.029	BLD - .178	.036	.069
Ethylbenzene	.066	BLD - .036	.005	.024
Xylenes	.022	BLD - .150	.025	.049
1,2,4-Trimethylbenzene	.017	BLD - .294	.009	.078
1,1,1-Trichloroethane	.036	BLD - .210	.061	.109
α-Pinene	.004	BLD - .033	.008	.022
Limonene	.005	BLD - .050	.006	.019
Ethanol	.170	BLD - 1.76	.239	.651
n-Octane	.005	BLD - 0.12	.011	-
n-Nonane	.037	BLD - .101	.084	-
n-Decane	.052	BLD - .137	.117	-

In all cases the distribution of observed hydrocarbon levels was not normal. Each case was positively skewed and leptokurtic. The exceptions to this is *n*-octane (-2.221), *n*-nonane (-1.781) and *n*-decane (-1.893) which had negative kutosis values, indicating a flatter platykurtic distribution.

The types of hydrocarbons found in each of the sampled buildings were similar to organic compounds found in most indoor environments, with the exception of two buildings (B9 and B10) which contained traces of aliphatic

FIGURE 8.1

Individual VOC concentrations in building one (weeks 1-10)



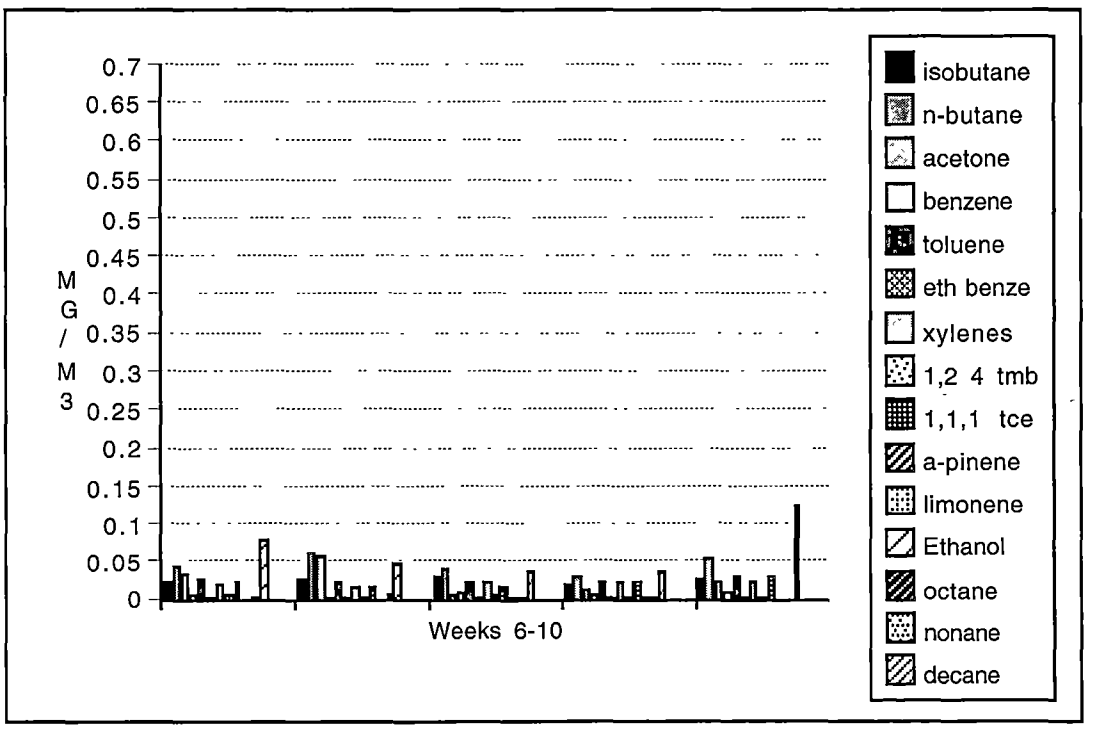
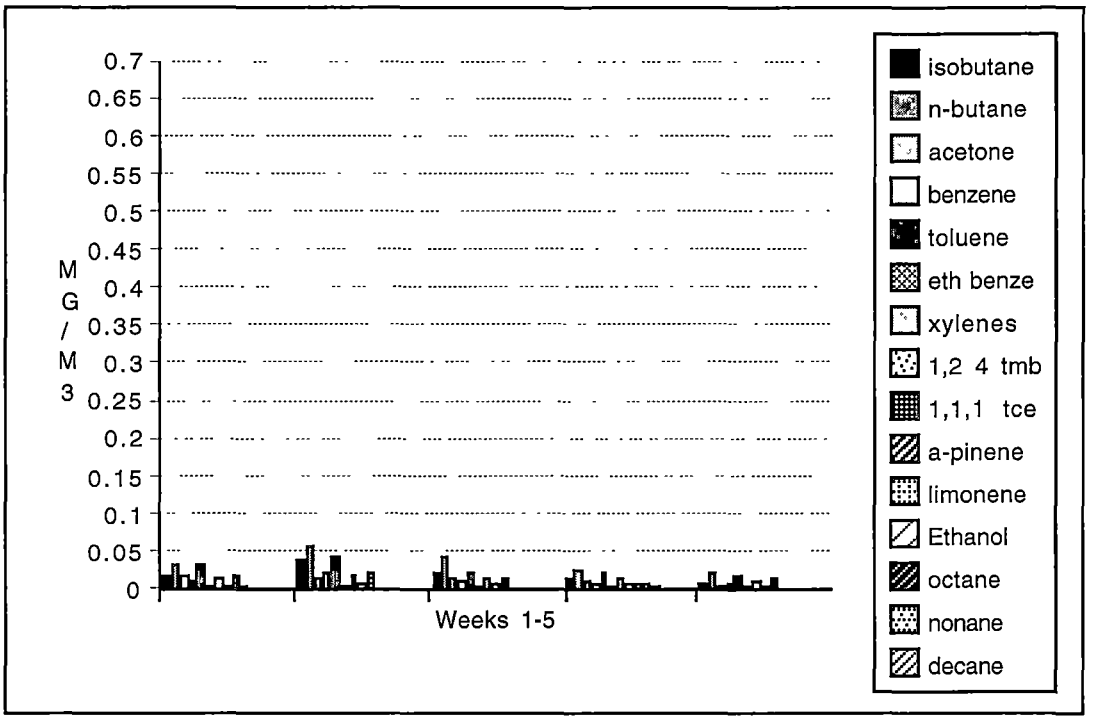


FIGURE 8.2

Individual VOC concentrations in building two (weeks 1-10)
Note: the ethanol levels represented in this graph exceed 0.7 mg/m^3. The actual value for ethanol week 4 was 1.15 mg/m^3



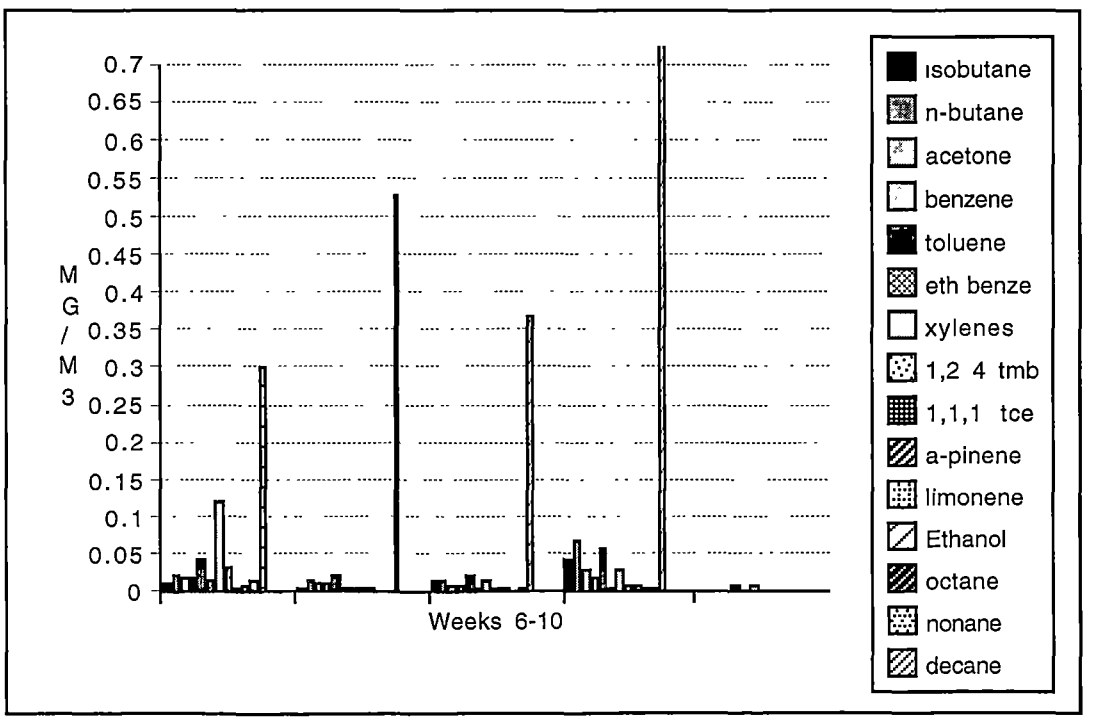
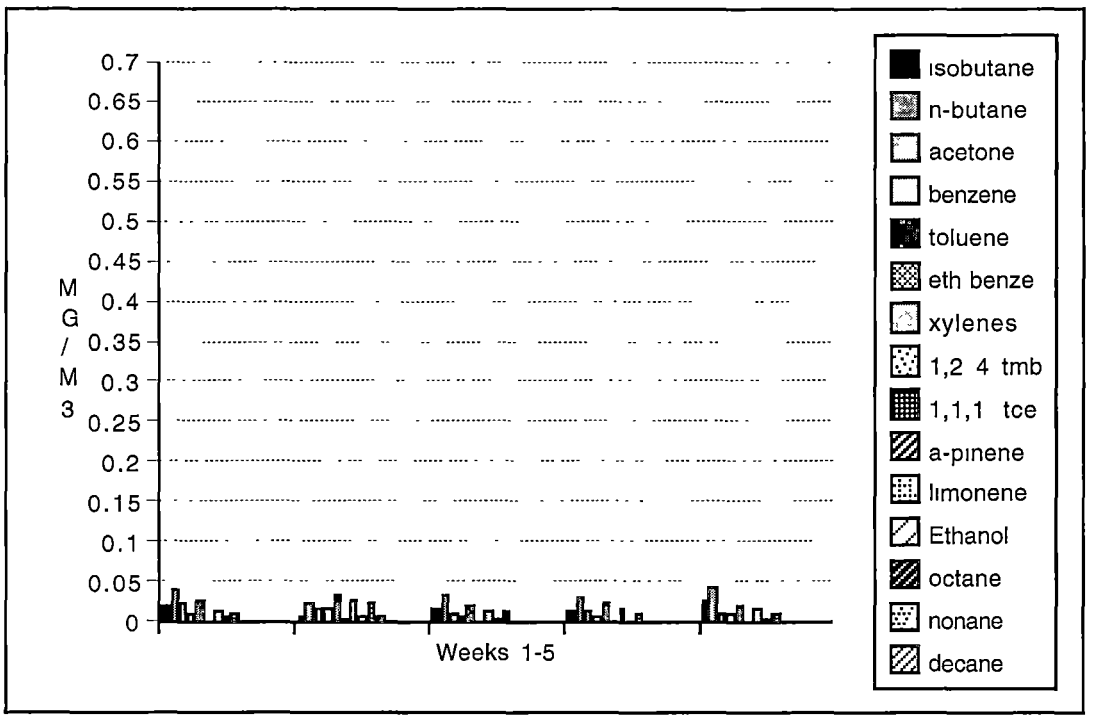


FIGURE 8.3

Individual VOC concentrations in building three (weeks 1-10)



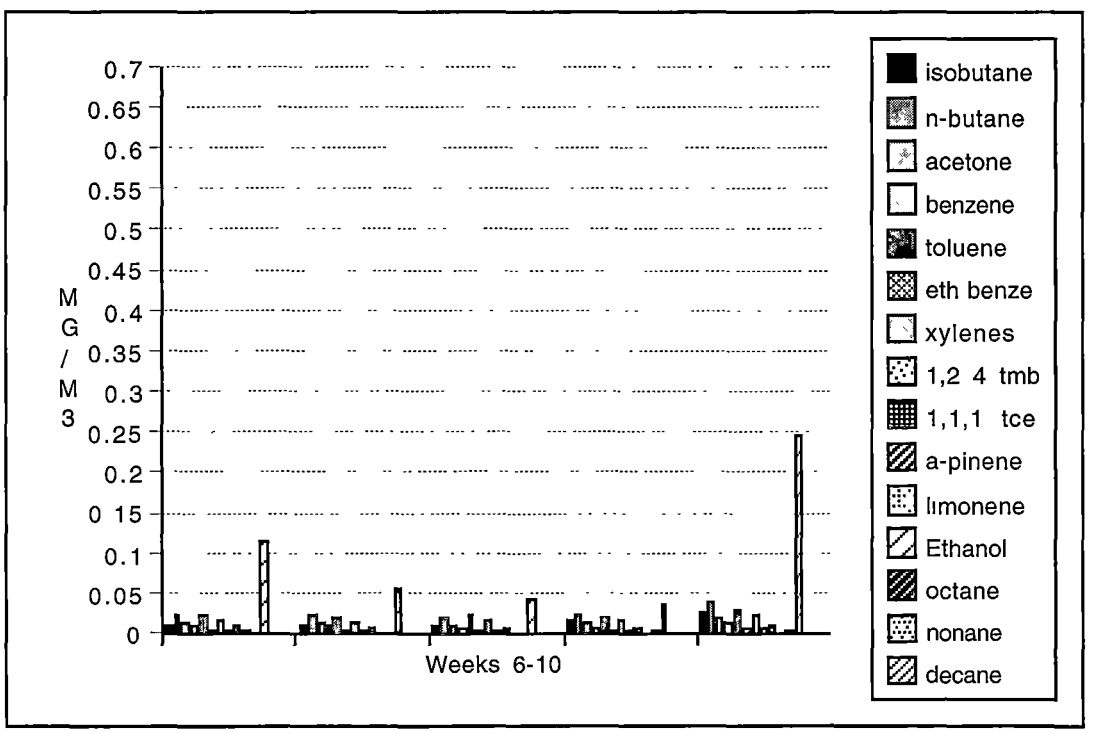
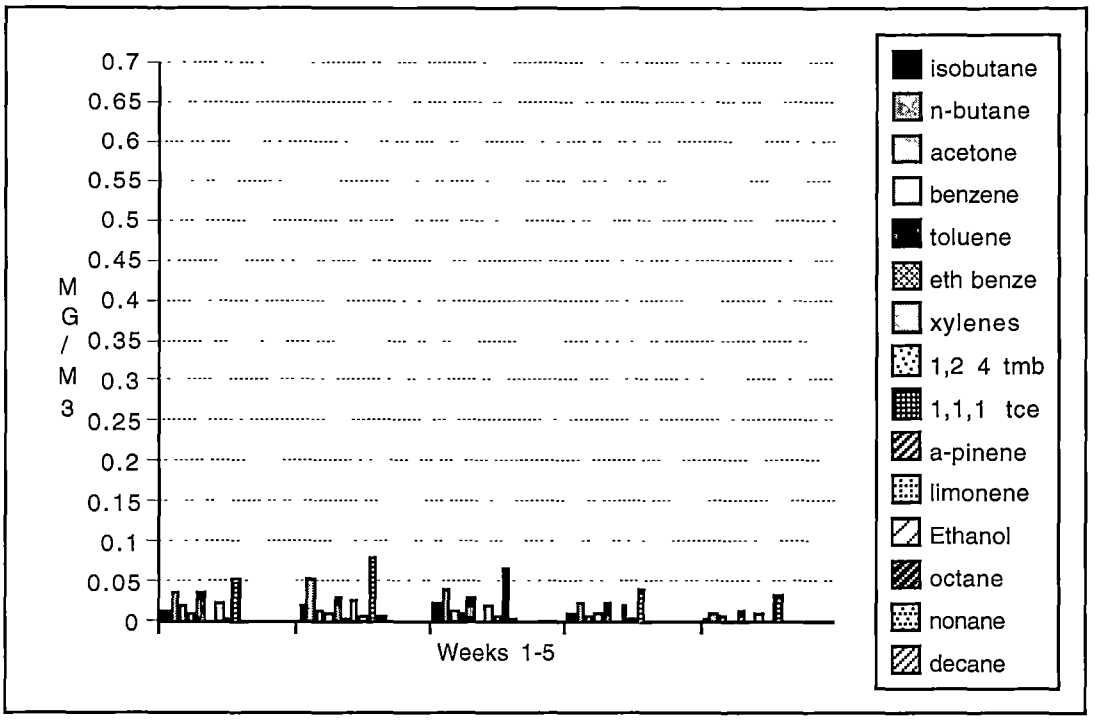


FIGURE 8.4

Individual VOC concentrations in building four (weeks 1-10)



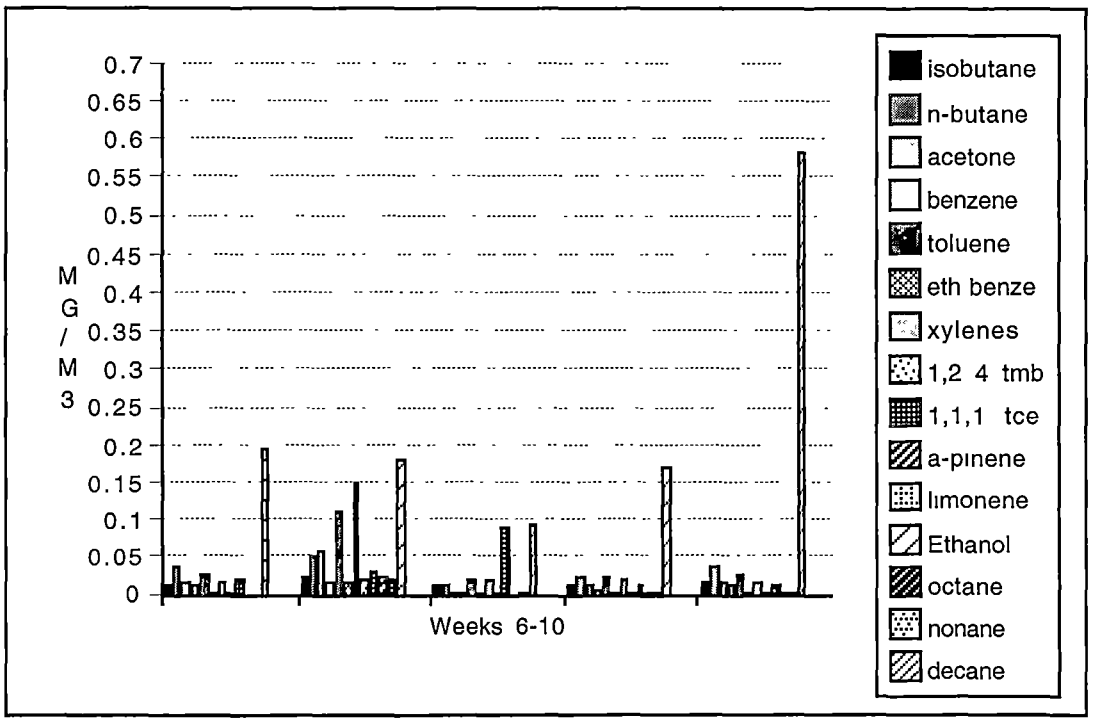
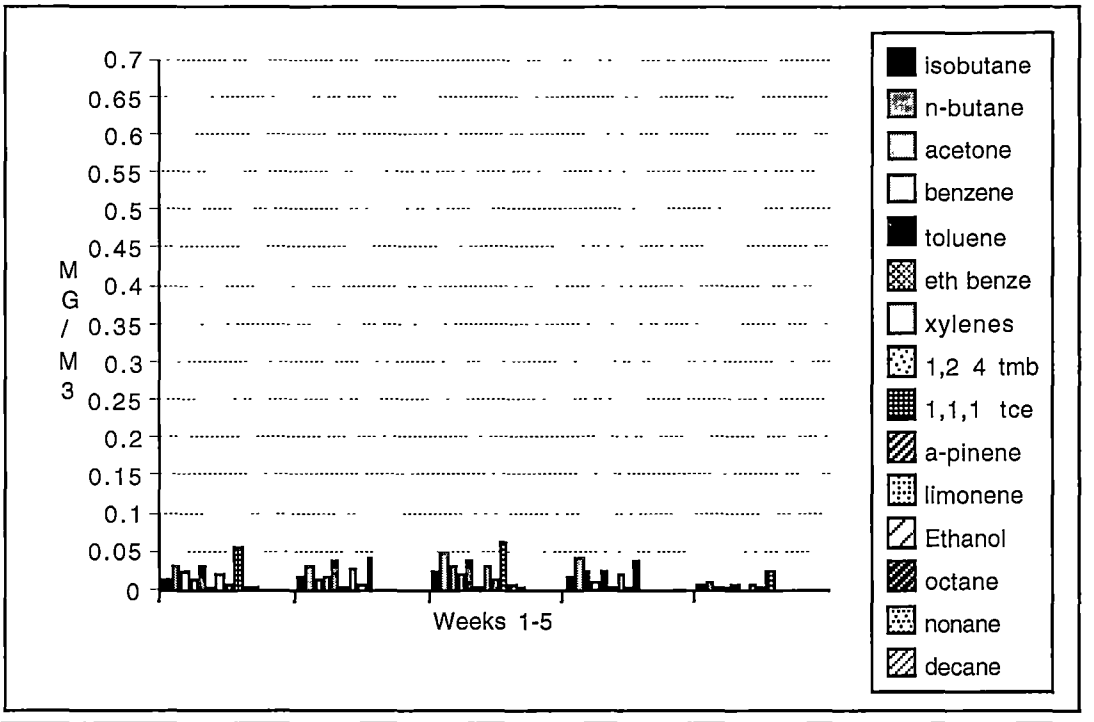


FIGURE 8.5

Individual VOC concentrations in building five (weeks 1-10)



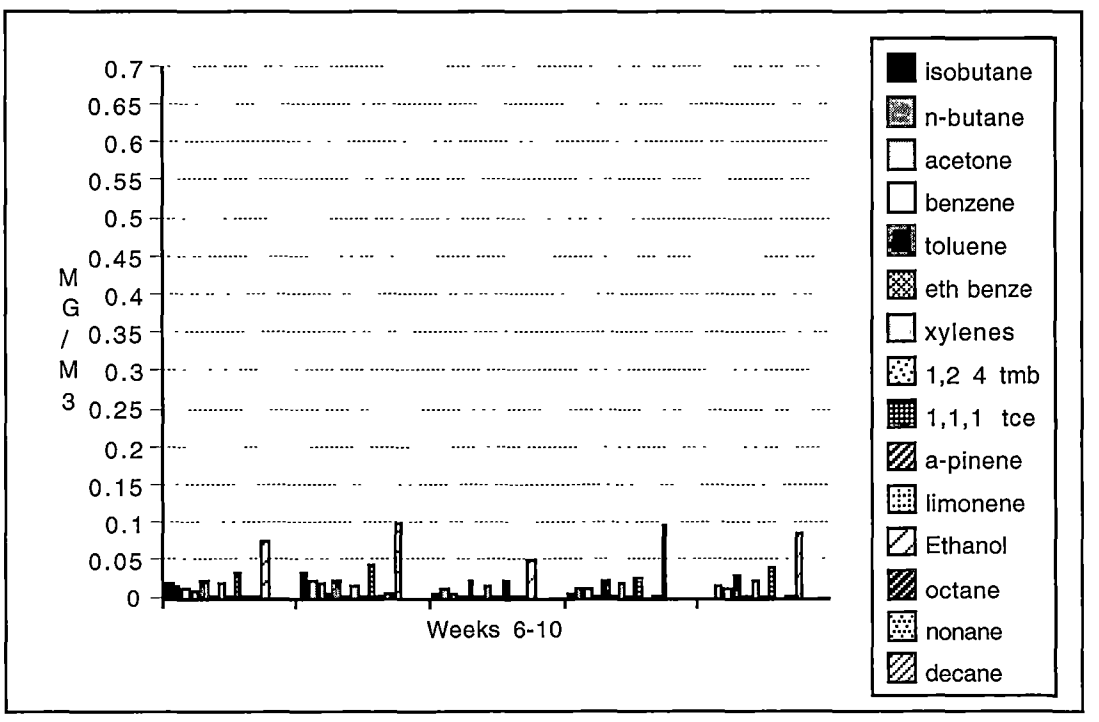
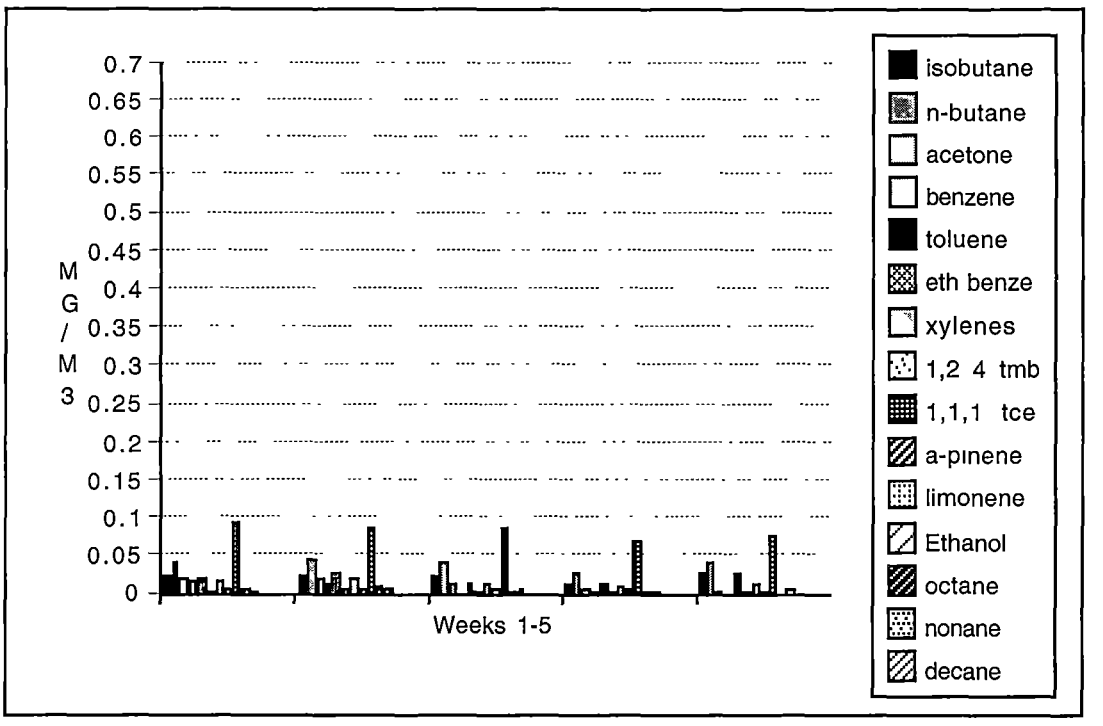


FIGURE 8.6

Individual VOC concentrations in building six (weeks 1-10)



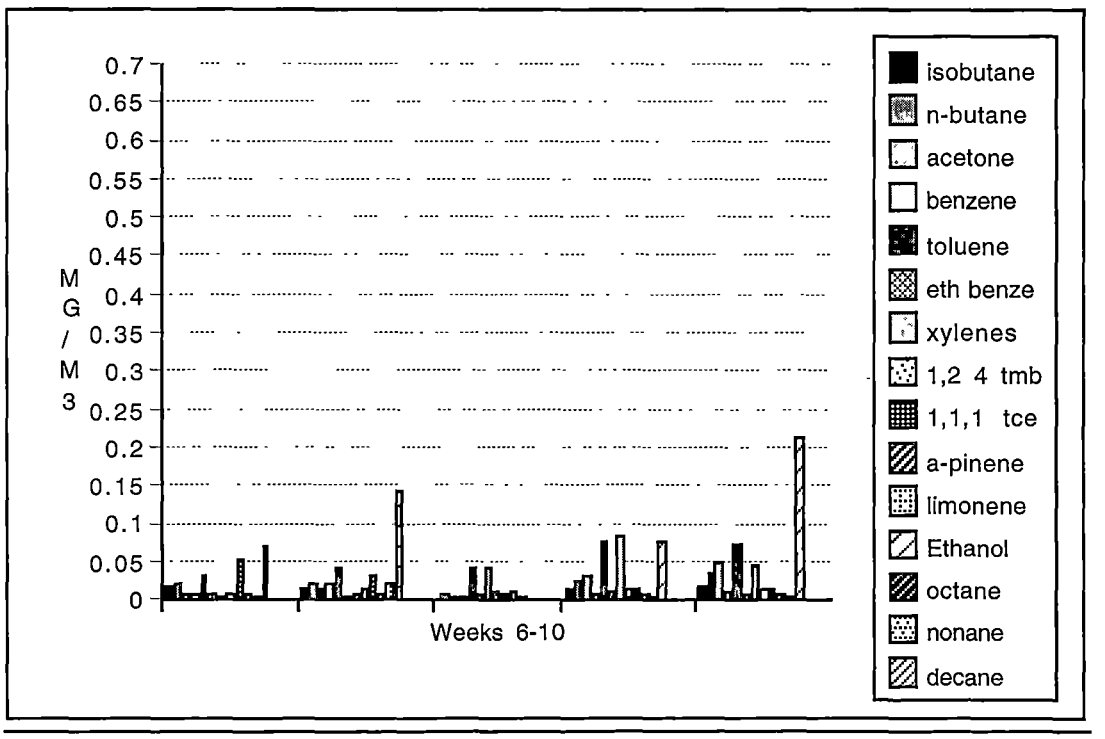
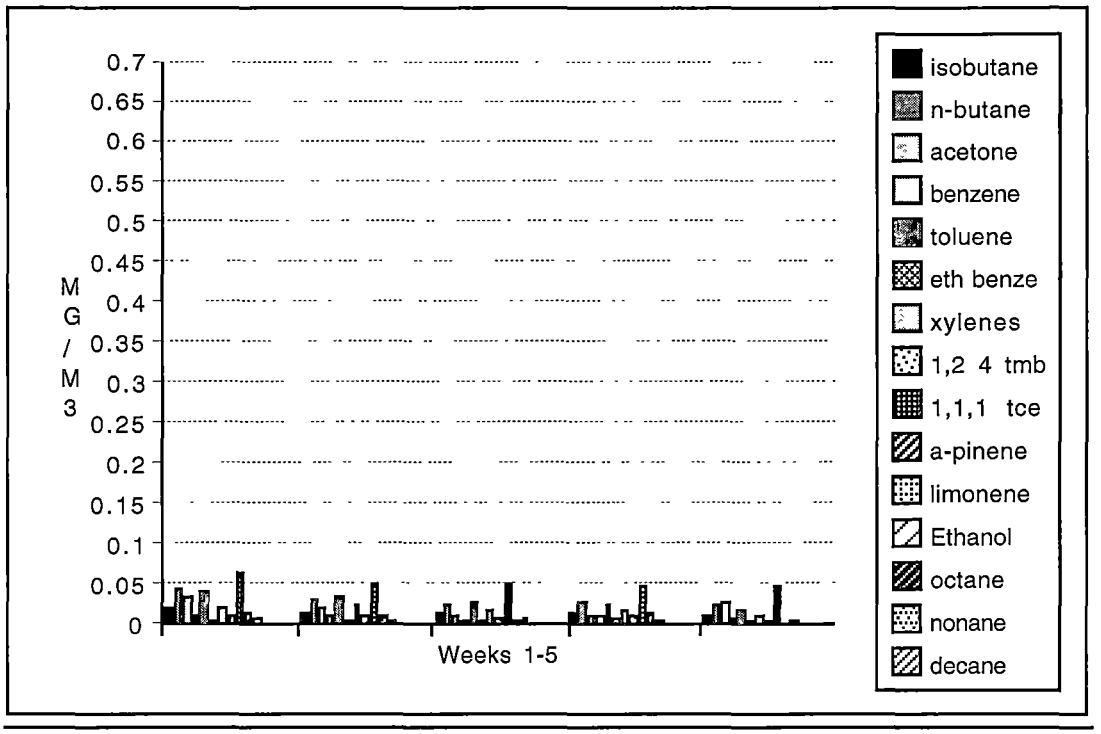


FIGURE 8.7

Individual VOC concentrations in building seven (weeks 1-10)



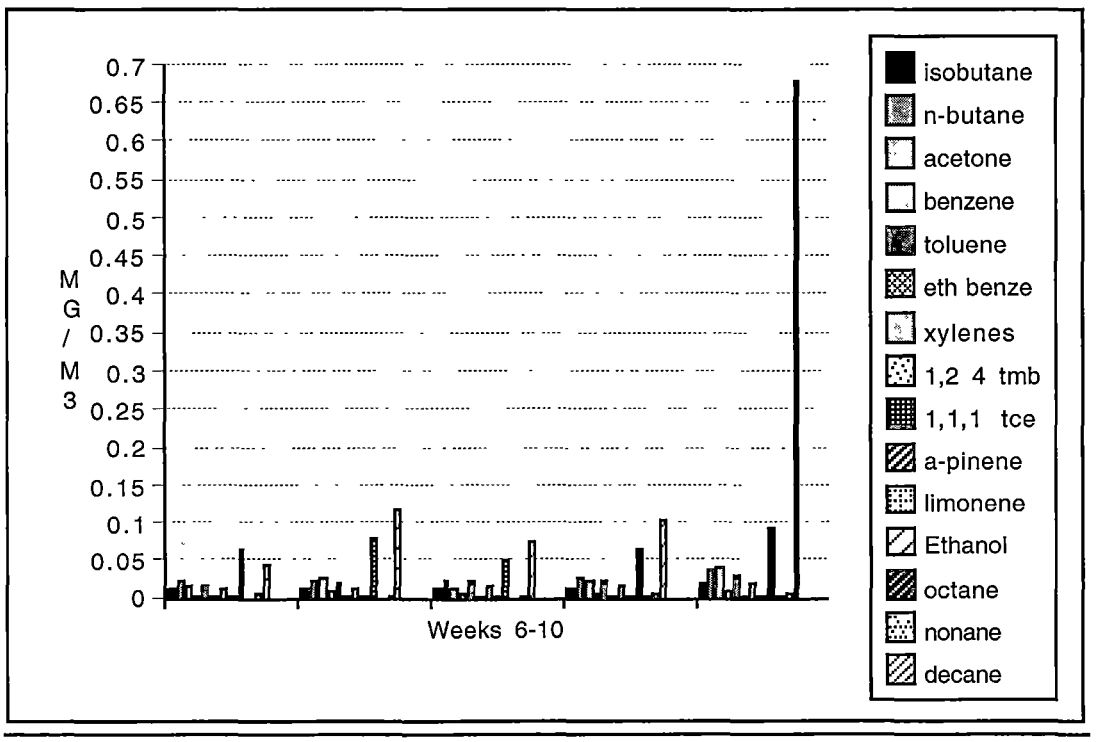
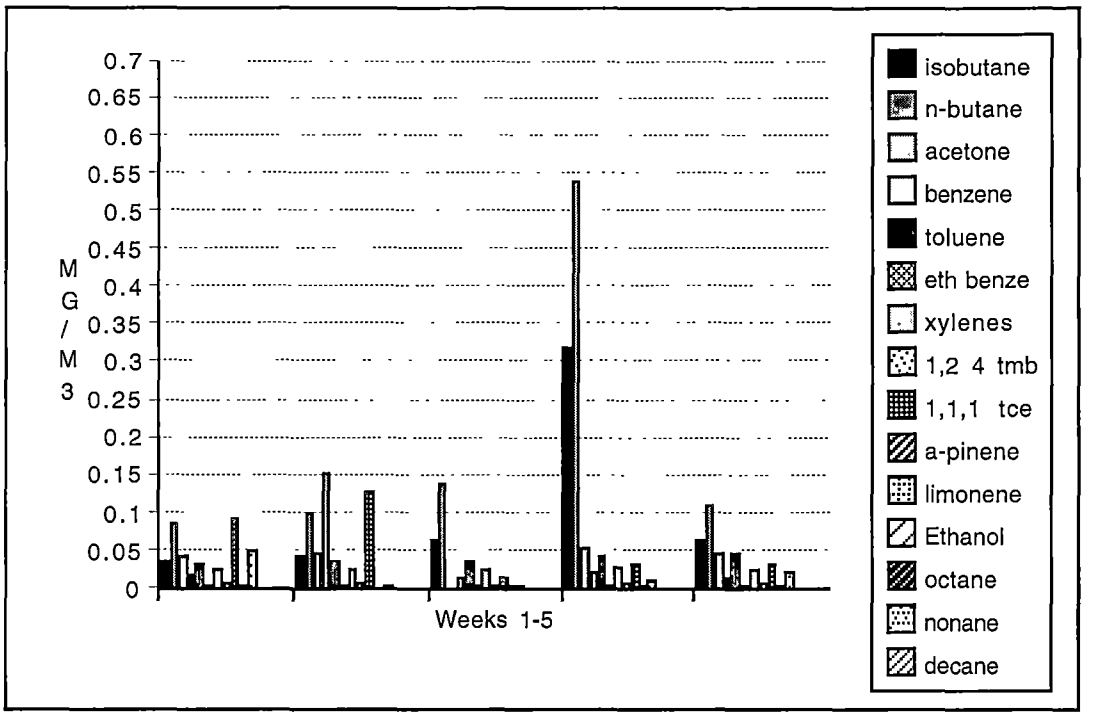


FIGURE 8.8

Individual VOC concentrations in building eight (weeks 1-10)



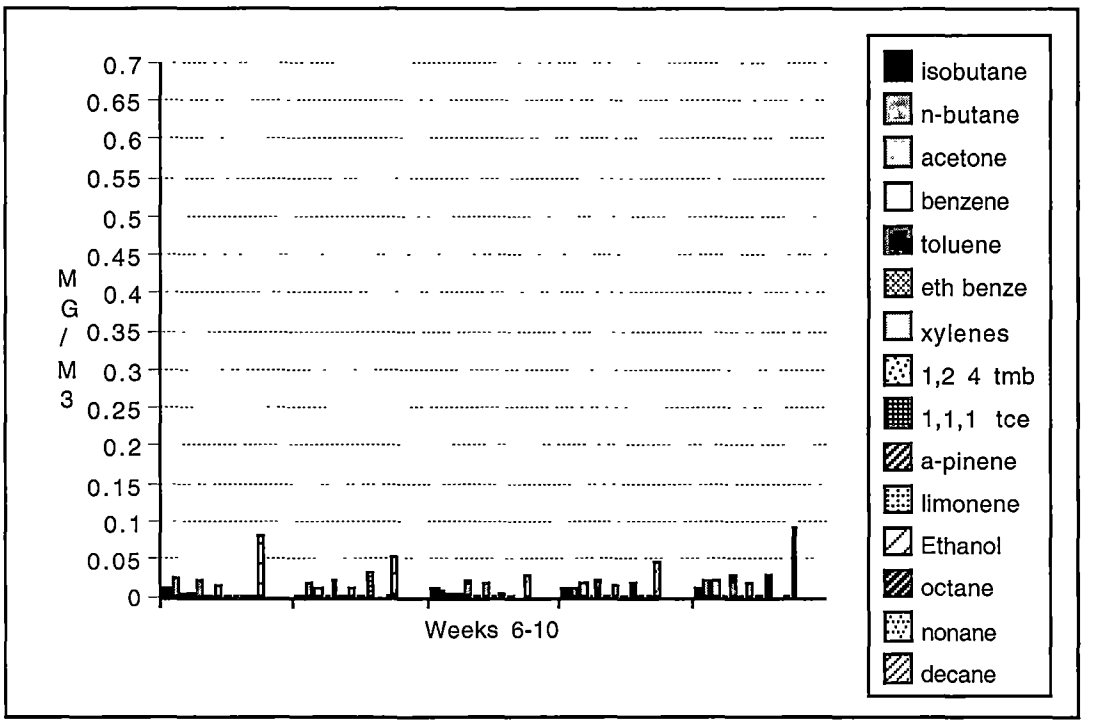
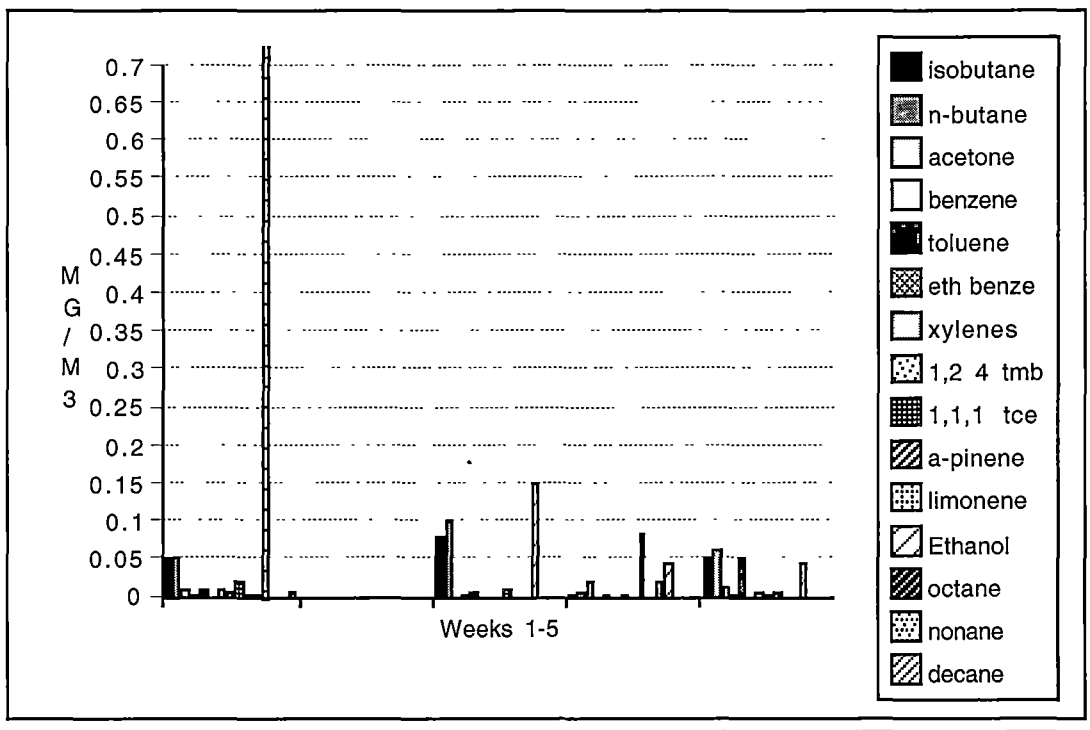


FIGURE 8.9

Individual VOC concentrations in building nine (weeks 1-10)
Note: the ethanol levels represented in this graph exceed 0.7 mg/m^3. The actual value for ethanol week 1 was 1.76mg/m^3



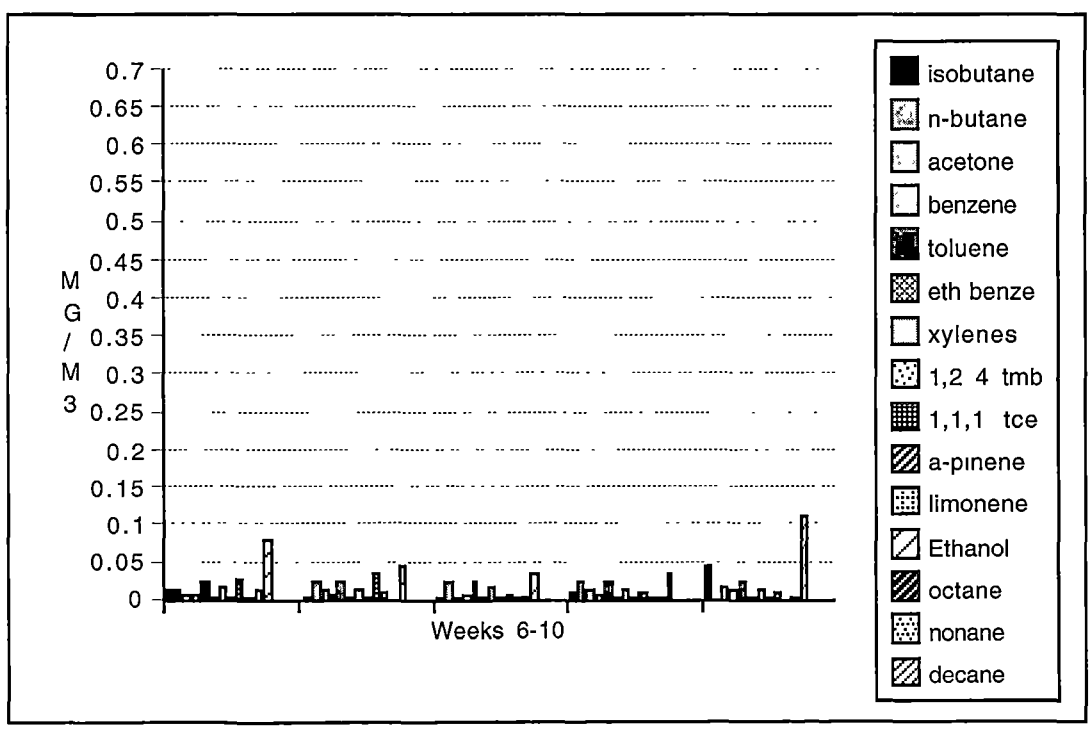
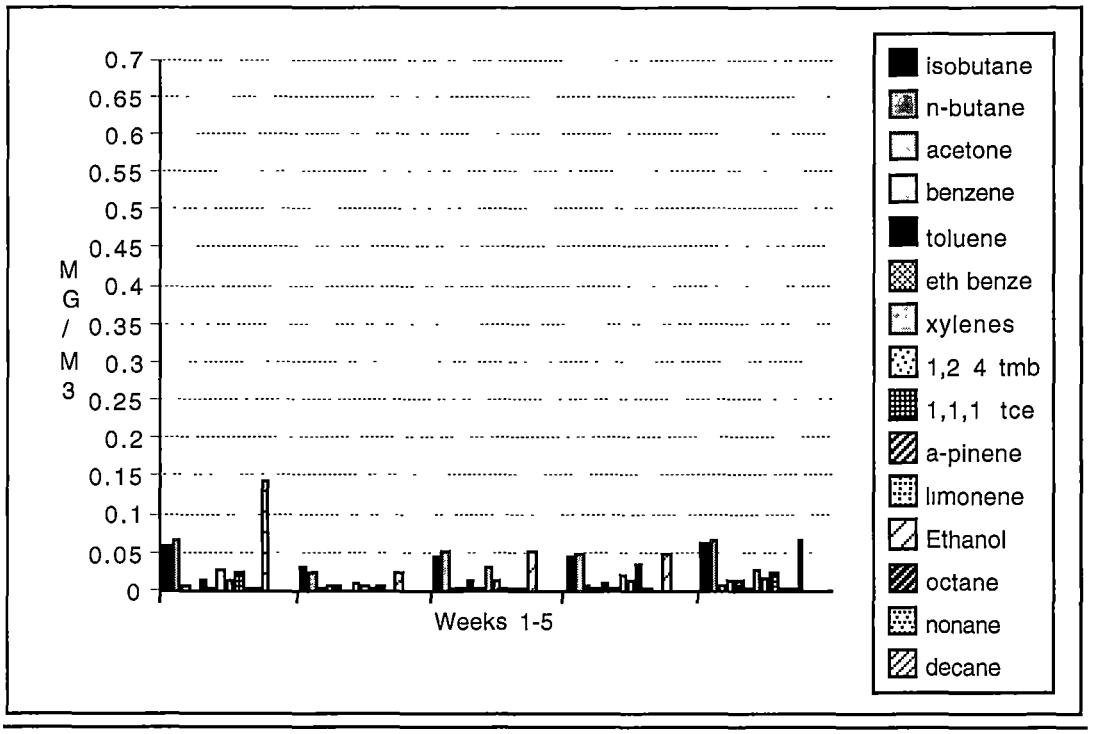


FIGURE 8.10

Individual VOC concentrations in building ten (weeks 1-10)



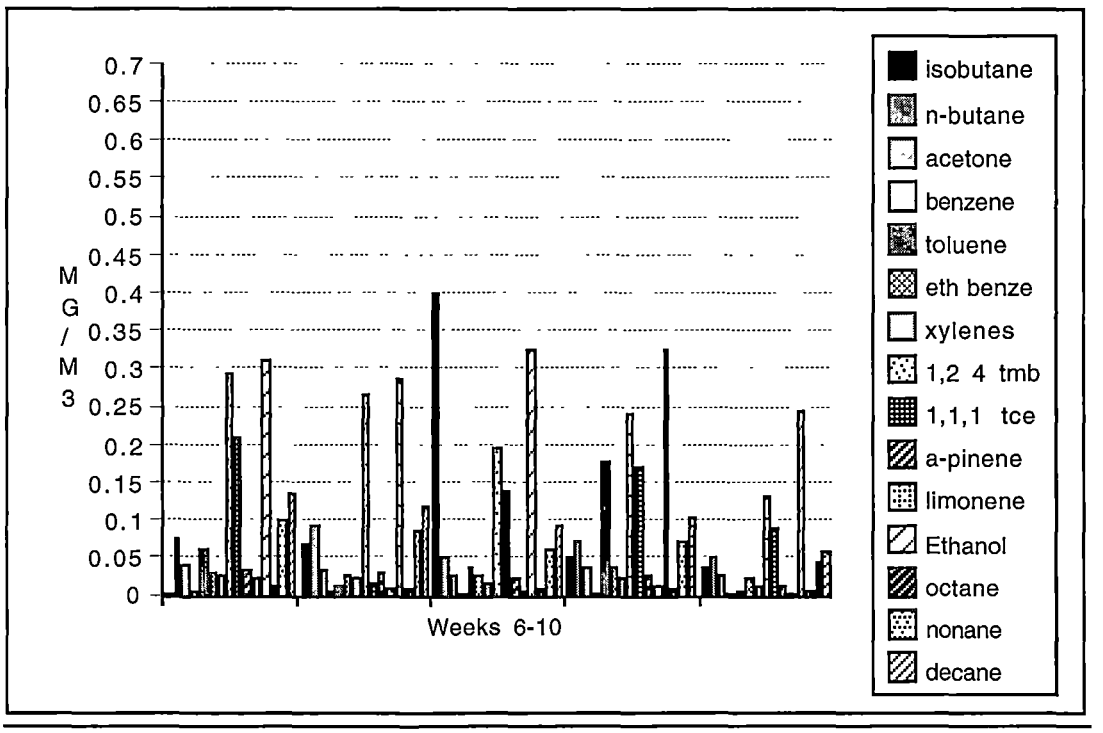
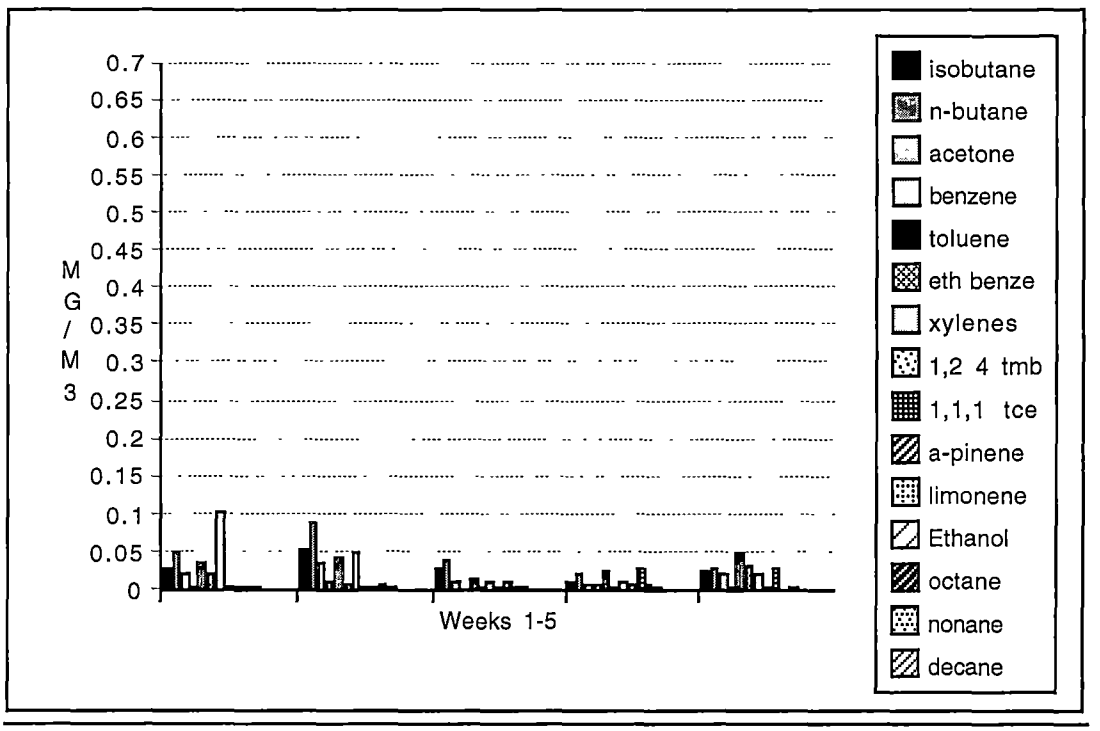


FIGURE 8.11

Individual VOC concentrations in building eleven (weeks 1-10)



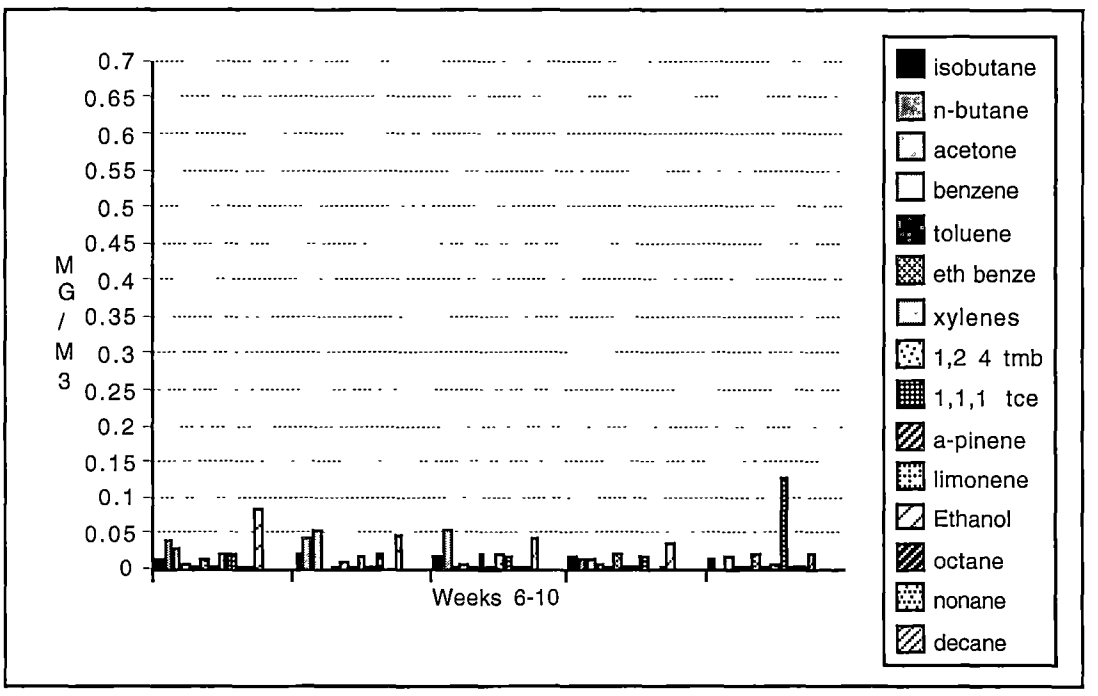
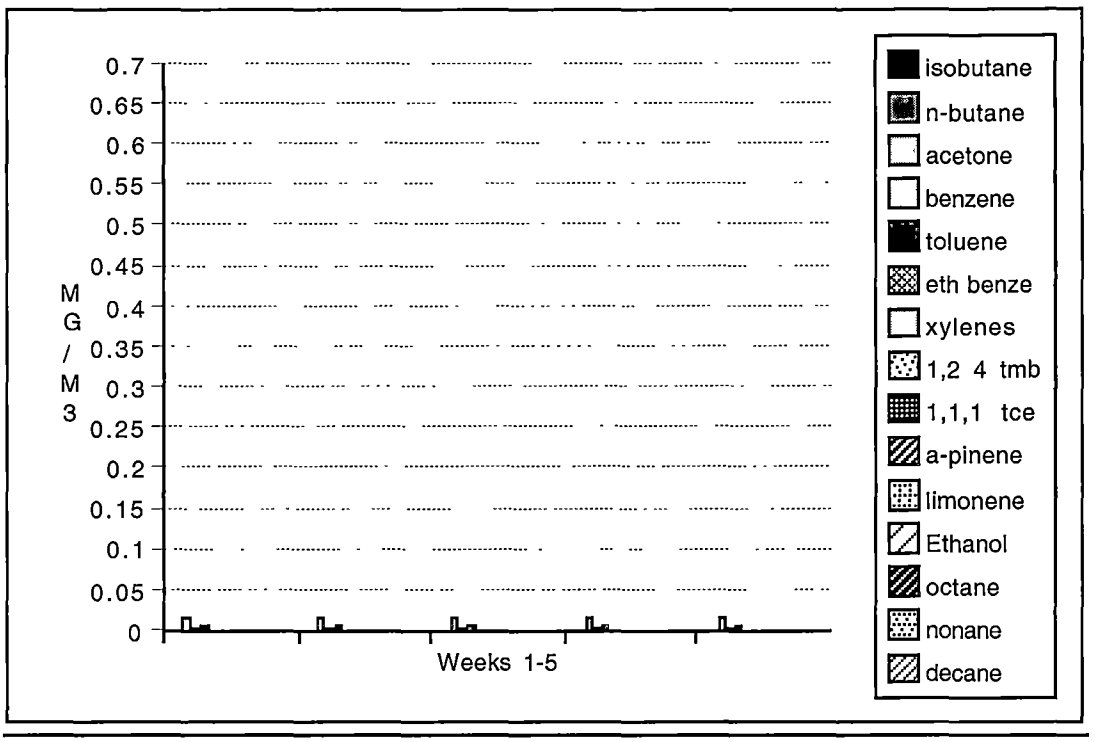
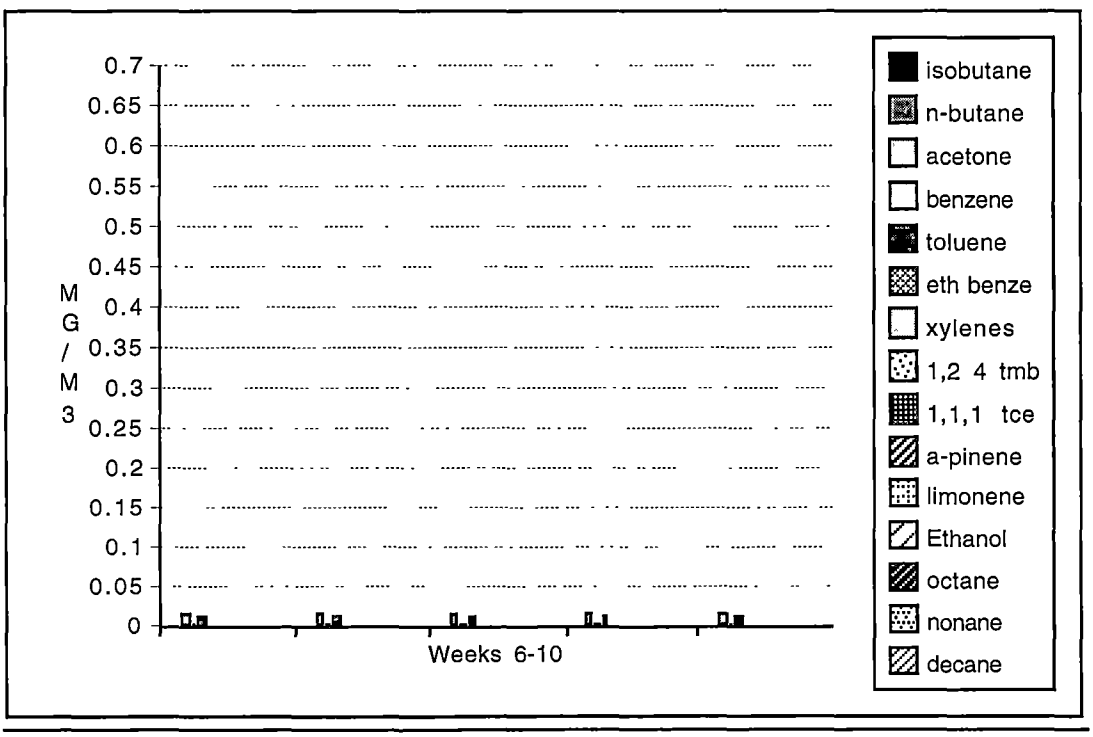


FIGURE 8.12

Individual VOC concentrations in the control building(weeks 1-10)





It appears from the graphical representation (figures 8.1 - 8.12) that increases in 1,1,1-trichloroethane are associated with elevated levels of ethanol. This was not the case in building B6, where 1,1,1-trichloroethane dominated measurable concentrations in weeks 1-5 and not weeks 6-10. Statistical analysis using the Spearman's Rank Order Correlation showed that ethanol was not correlated with 1,1,1-trichloroethane (>.05). Instead, levels of ethanol were associated with *n*-decane (<.05) (figure 8.16).

Interestingly, further investigations revealed that as 1,1,1-trichloroethane concentrations increased so did the levels of 1,2,4-trimethylbenzene, acetone, *n*-decane, *n*-nonane, *n*-octane, and limonene (<.05).

Generally building B10 had the most extensive range of hydrocarbons, with weeks 6-10 displaying some of the highest concentrations detected during the sampling period. Building B8 had a peak in isobutane and *n*-butane during week four, in contrast to building B1 which showed a more consistent elevation of isobutane and *n*-butane.

It appears that structurally related compounds have a strong association with one another. This is illustrated in the graphical representations of the building hydrocarbon levels. Compounds with the same molecular formula but different structural formulas, or isomers, are more likely to be present in combination with one another rather than individually. For example, higher levels of isobutane are associated with higher levels of *n*-butane. This was found to be statistically significant using the Spearman's Rank Order Coefficient (<.05). The most reasonable explanation for this result is that either there is a structural similarity between the compounds or an association in a common source.

8.3 Total Volatile Organic Compound Concentrations (TVOC)

As seen in table 8.2, TVOC levels in the eleven sampled buildings ranged from 0.012-1.934 mg/m^3. The concentrations in the control building ranged from 0.025-0.029 mg/m^3. As with the individual VOC values, TVOC levels are 7 day averages.

Generally, the TVOC levels remained fairly consistent during the ten sampling weeks, except for a few isolated cases (B2 - week 9, B8 - week 4, B9 - week 1 and B10 - weeks 6,7,8,9). These increases in TVOC levels may be associated with a particular building event such as cleaning regimes or factors external to the indoor environment.

FIGURE 8.13

Scattergram displaying the correlation bewteen *n*-butane and acetone

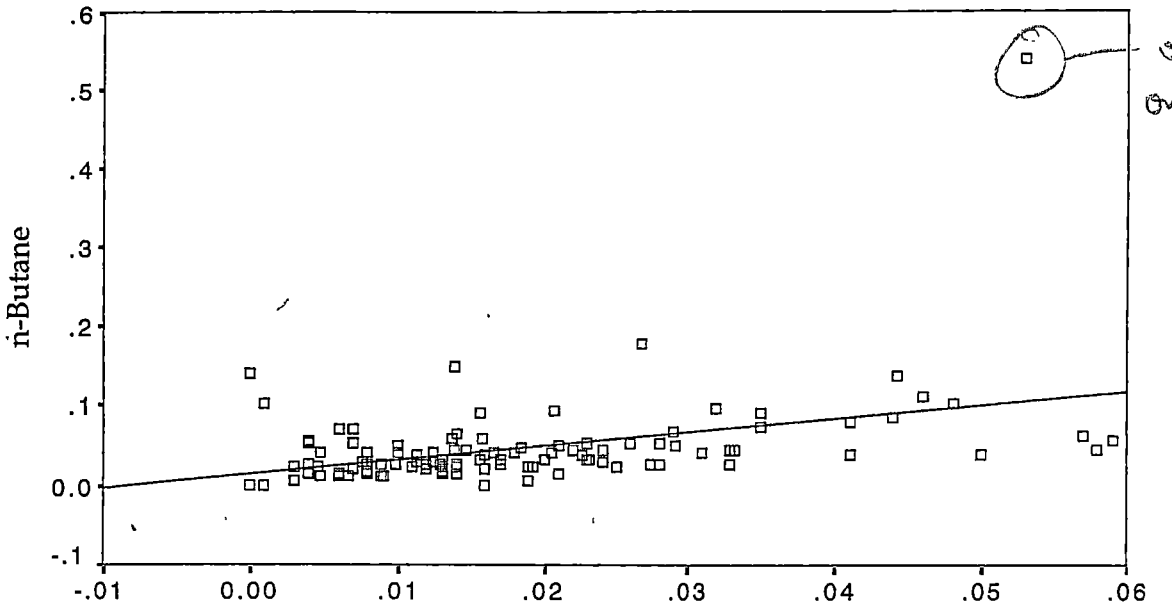


FIGURE 8.14

Scattergram displaying the correlation bewteen *n*-butane and toluene

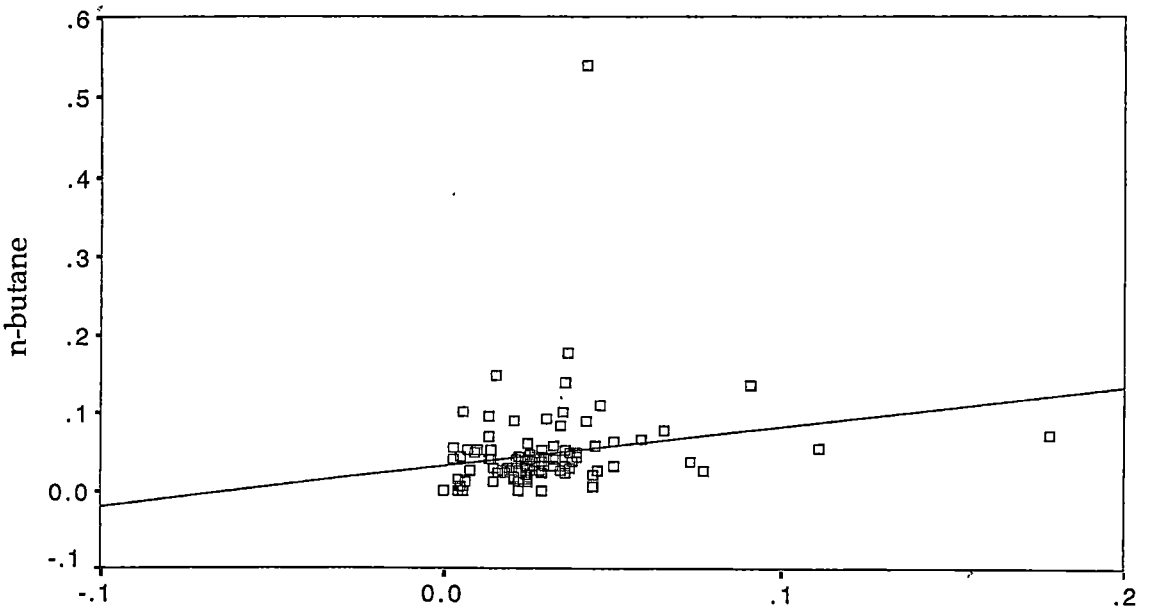


FIGURE 8.15

Scattergram displaying the correlation bewteen *n*-butane and xylene

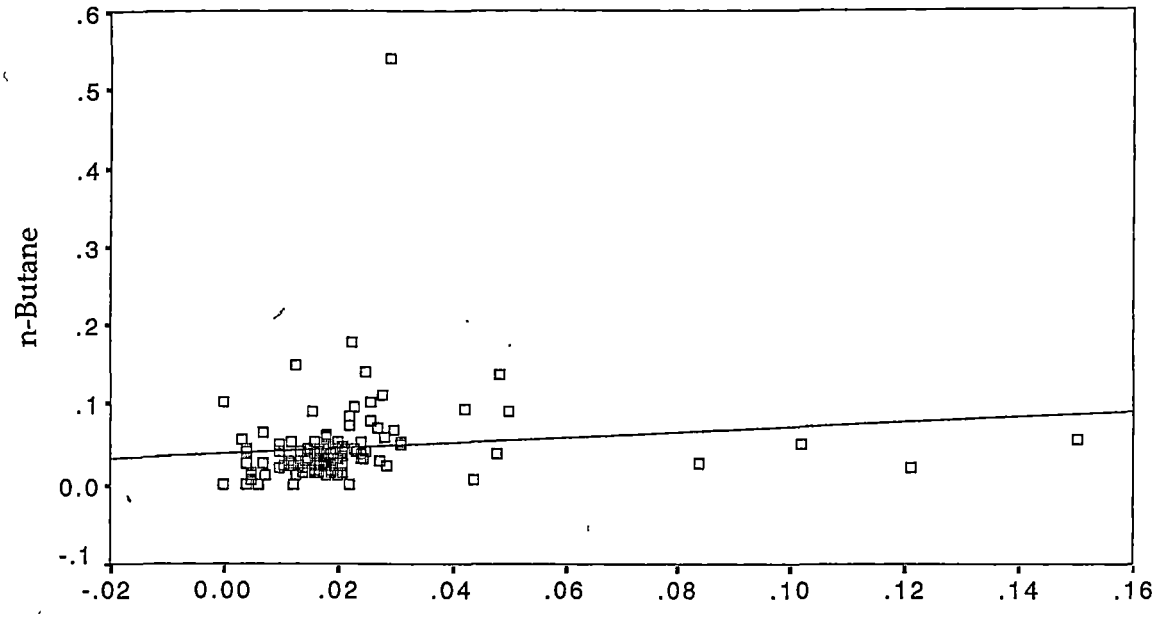
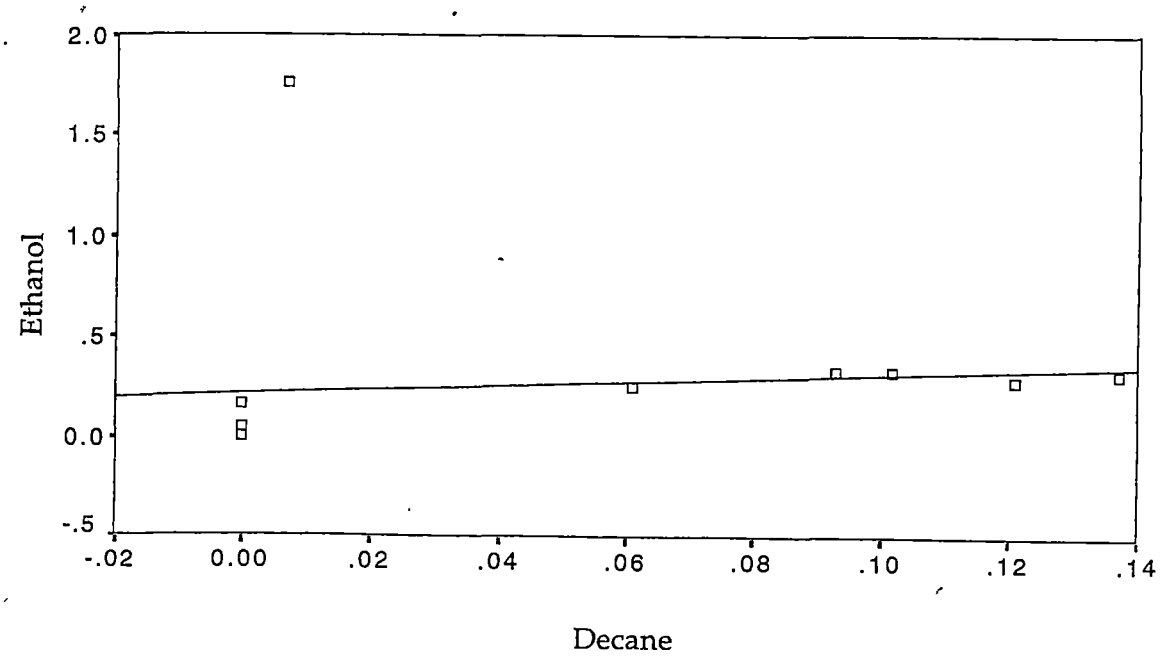


FIGURE 8.16

Scattergram displaying the correlation between ethanol and *n*-decane



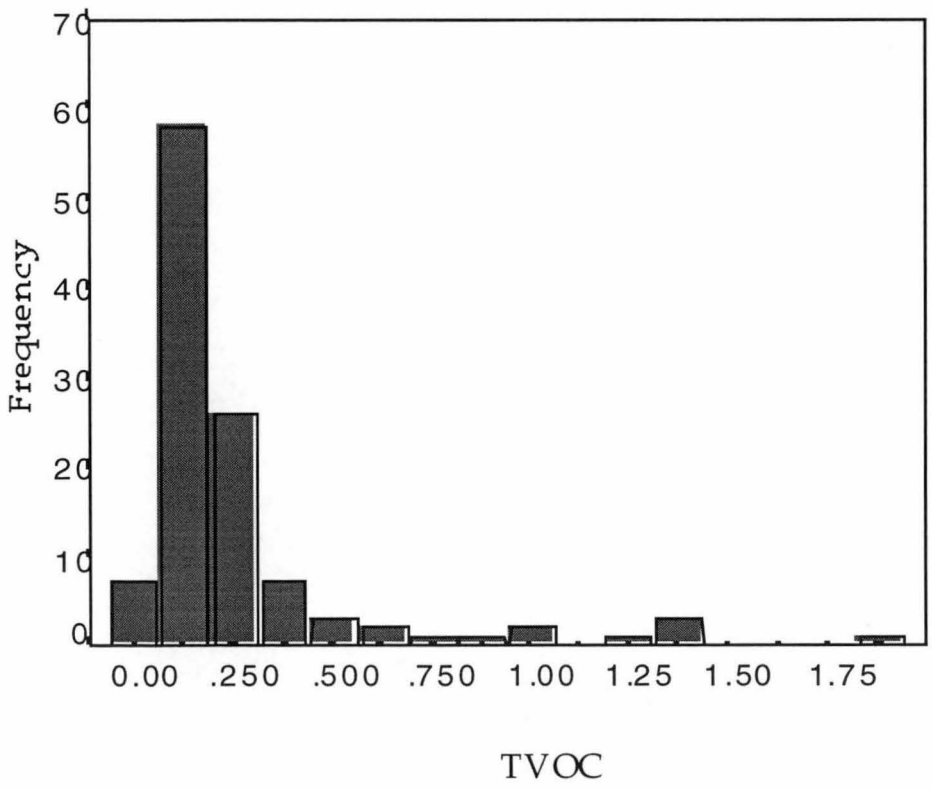
For example, building B1 had a considerable rise in TVOC levels in week 5 of sampling after steam cleaning of carpets. Similarly, buildings with high TVOC levels (B4, B8, B9 and B10) all had refurbishing practices undertaken within the last five years. It is possible that materials used in the refurbishment process are still outgassing, as would be the case with paints and carpets.

The Spearman's Rank Order Correlation showed that building population numbers in each of the sampled buildings had no significant effect on TVOC concentrations. Results were well above the alpha level of .05.

Figure 8.17 illustrates the TVOC concentration values determined in the eleven buildings. The high concentrations at the end of the distribution (> 1.50 mg/m^3) could be partially due to chemicals commonly used in offices such as 1,1,1 trichloroethane from correction fluids or the infiltration of heavily contaminated air, e.g. in close proximity to areas of high vehicular traffic.

FIGURE 8.17

Distribution of TVOC (mg/m^3) concentration values in the eleven sampled buildings and the control



Although data resulting from the methodology used was not readily interpretable into excursions during 1hr, it was still possible to refer the VOC/TVOC standards such as the NHMRC goal of 500 $\mu g/m^3$ to the results.

As seen in table 8.2, the mean TVOC concentrations (over 7 sampling days) exceeded the NHMRC one hour goal of 500 $\mu g/m^3$ on fourteen occassions. Although what is not known is whether these TVOC values were steady during the seven day sampling period or whether they peaked at some time during the week and then maintained a low value for the remainder. It is evident though from the results that some, or all, of the one hour periods must have exceeded 500 $\mu g/m^3$.

TABLE 8.2

TVOC levels (mg/m^3) for sampled buildings, weeks 1-10 and a control

No	W1	W2	W3	W4	W5	W6	W7	W8	W9	W10
B1	0.262	0.438	0.310	0.291	0.489	0.278	0.269	0.208	0.193	0.330
B2	0.149	0.224	0.151	0.120	0.085	0.603	0.603	0.459	1.424	0.012
B3	0.166	0.187	0.139	0.141	0.164	0.229	0.151	0.135	0.151	0.421
B4	0.218	0.271	0.240	0.156	0.096	0.394	0.712	0.269	0.299	0.736
B5	0.206	0.194	0.283	0.184	0.065	0.228	0.282	0.156	0.222	0.221
B6	0.251	0.260	0.207	0.165	0.201	0.246	0.356	0.138	0.372	0.498
B7	0.277	0.213	0.170	0.186	0.155	0.206	0.316	0.231	0.297	0.945
B8	0.392	0.548	0.308	1.062	0.374	0.188	0.176	0.126	0.171	0.250
B9	1.934	0.012	0.352	0.190	0.240	0.210	0.178	0.130	0.147	0.242
B10	0.366	0.125	0.228	0.245	0.309	1.378	1.091	1.418	1.367	0.760
B11	0.277	0.302	0.130	0.132	0.222	0.245	0.233	0.200	0.143	0.224
BC	0.025	0.025	0.025	0.025	0.025	0.029	0.029	0.029	0.029	0.029

Legend

TVOC concentrations over the NHMRC goal of 500 $\mu g/m^3$ (1 hour average)

TVOC concentrations slightly below the NHMRC goal of 500 $\mu g/m^3$ (1 hour average)

Building B10 was the worst with concentrations exceeding the 500 $\mu g/m^3$ for five out of the ten sampling weeks, followed by building B2 which had concentrations exceeding the goal for three out of the ten sampling weeks. Building B9 had the

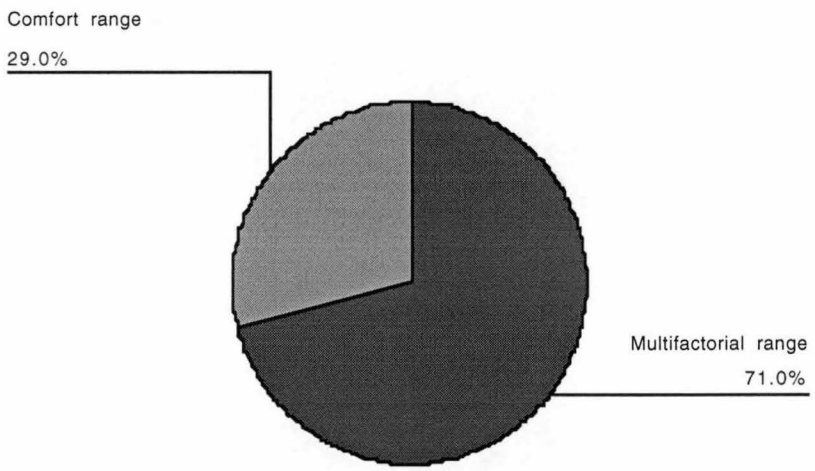
highest TVOC of 1934 $\mu g/m^3$, with buildings B10 (1418 $\mu g/m^3$) and B2 (1424 $\mu g/m^3$) at fairly high TVOC levels.

It is also likely that the concentration measured during one hour intervals would not be constant over one week. Some values would have peaked above the weekly average, and others below. Therefore, if the weekly average concentration is slightly below the one hour goal of 500 $\mu g/m^3$, it would be very likely that the goal was exceeded at some time during the week (as seen in table 8.2). This is illustrated in the results where on fifteen occassions the goal was almost exceeded. Overall, the buildings that had concentrations close to the NHMRC goal are the same buildings that had concentrations which in some weeks actually exceeded the 500 $\mu g/m^3$ during the seven sampling days (e.g. building B10, B9, B8, B7, B4, and B2).

As seen in figure 8.18, 29% of the sampled buildings were under the TVOC comfort range (< .02 mg/m^3), while 71% were within the range of 0.20 - 3.0 mg/m^3 which is thought to cause possible irritation and discomfort (Molhave 1995).

FIGURE 8.18

Percentage of buildings in the comfort or multifactorial exposure range



8.3.1 WEEKLY VARIATIONS IN CONCENTRATIONS

As seen in figures 8.19 - 8.30, the weekly TVOCs show similar concentration values in weeks 1 through to ten. In week two 63.6% of buildings had a noticeable rise in TVOCs, whereas weeks 3-4 are in the majority of cases lower than weeks 1, 2 and 5. Generally weeks 6 and 7 of sampling have equal TVOC levels until week ten where 82% of buildings show a significant increase in TVOC concentrations.

FIGURE 8.19

TVOC levels (mg/m^3) for building one (weeks 1-10)

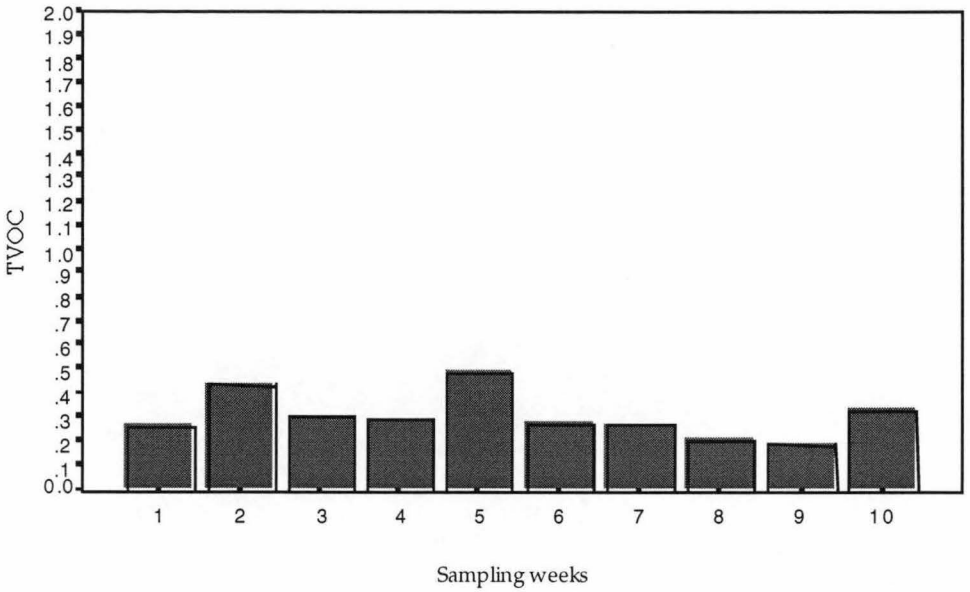


FIGURE 8.20

TVOC levels (mg/m^3) for building two (weeks 1-10)

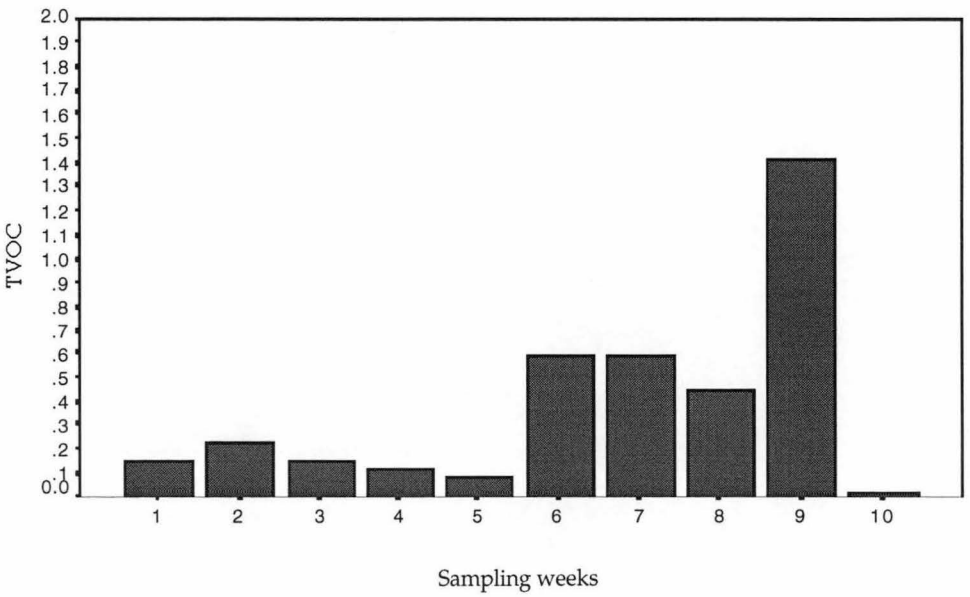


FIGURE 8.21

TVOC levels (mg/m^3) for building three (weeks 1-10)

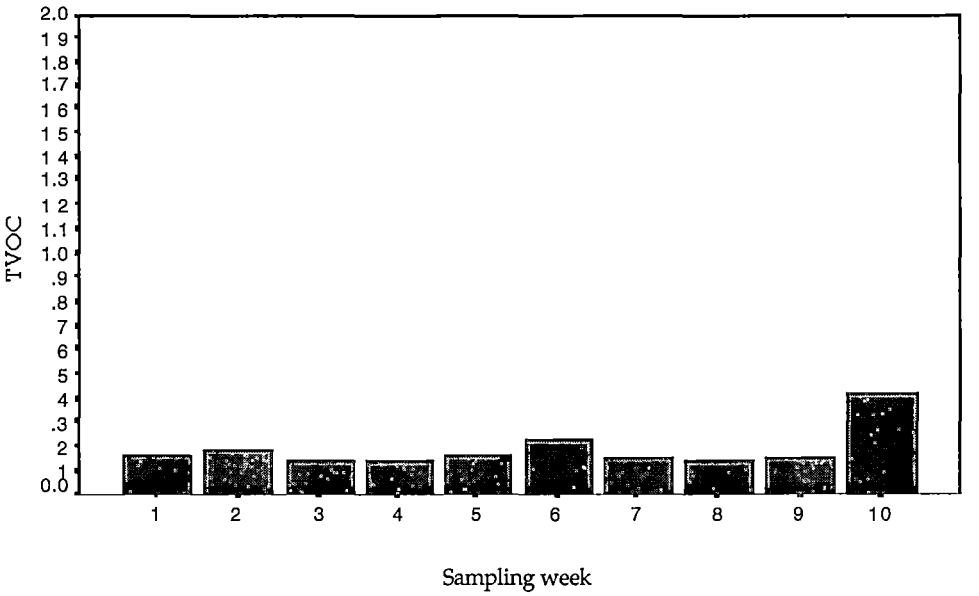


FIGURE 8.22

TVOC levels (mg/m^3) for building four (weeks 1-10)

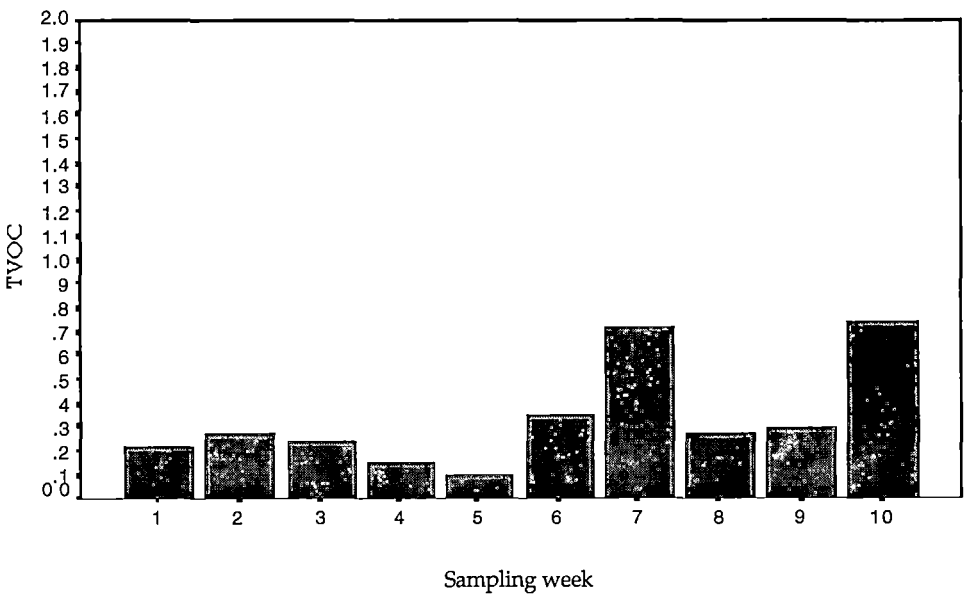


FIGURE 8.23

TVOC levels (mg/m^3) for building five (weeks 1-10)

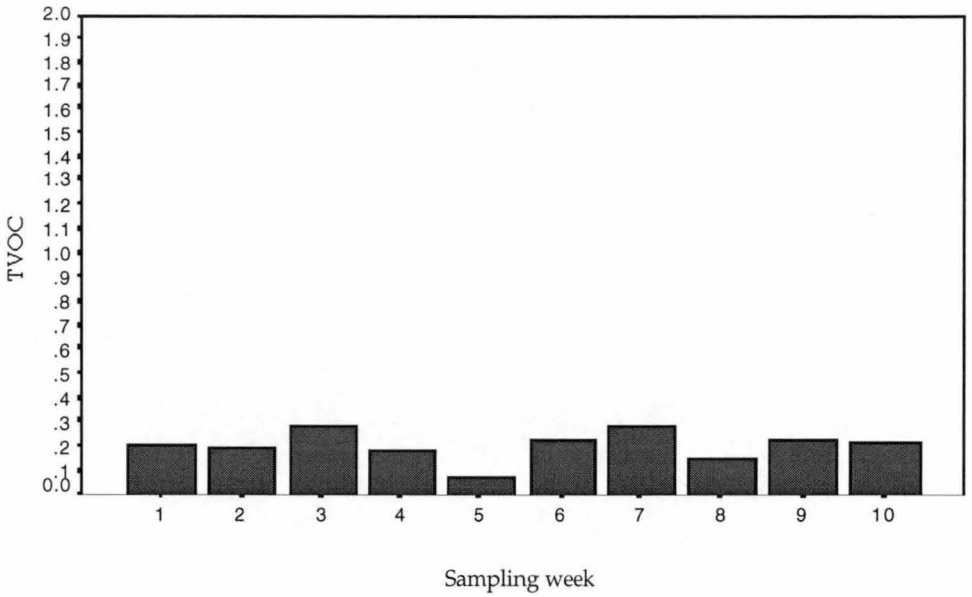


FIGURE 8.24

TVOC levels (mg/m^3) for building six (weeks 1-10)

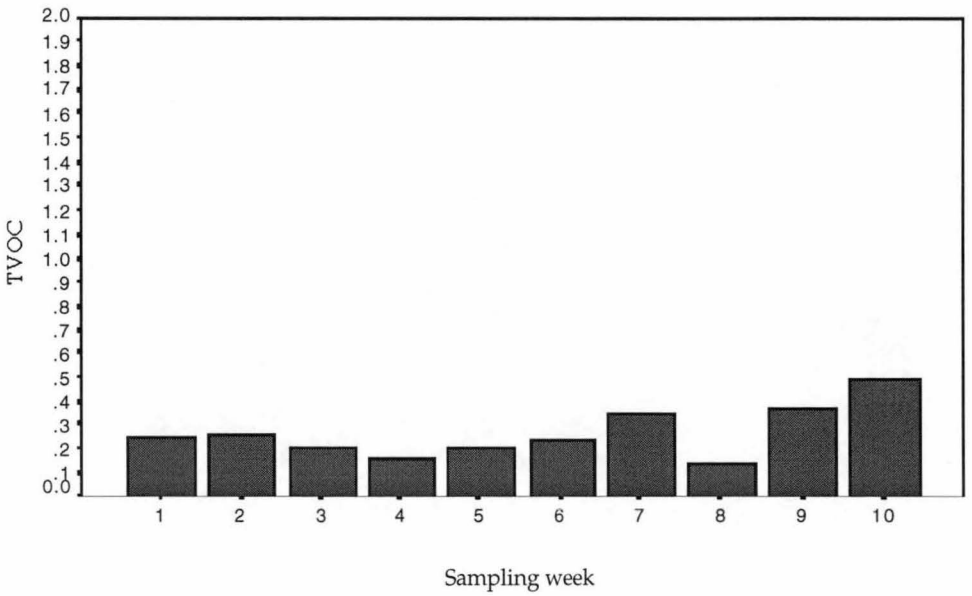


FIGURE 8.25

TVOC levels (mg/m^3) for building seven (weeks 1-10)

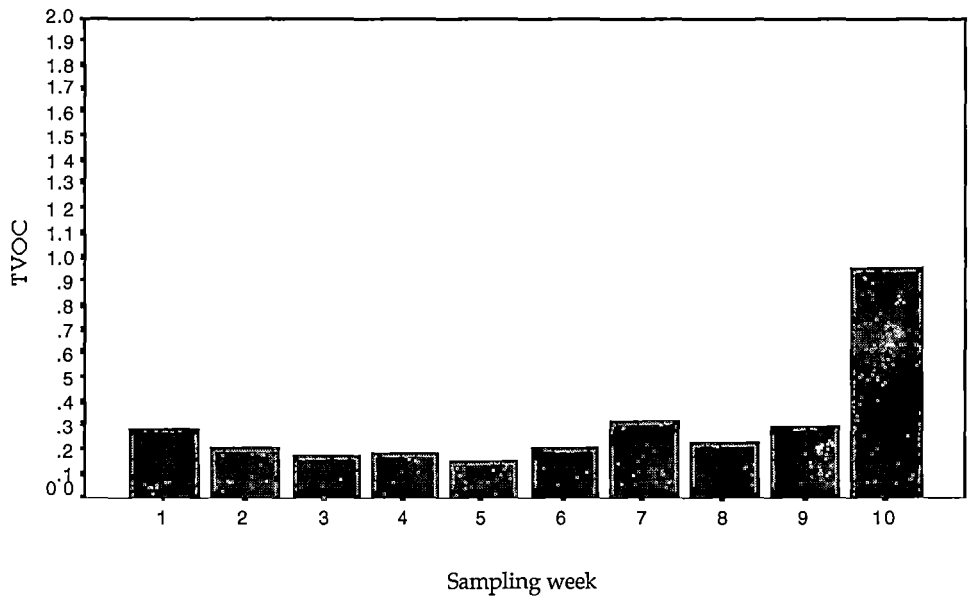


FIGURE 8.26

TVOC levels (mg/m^3) for building eight (weeks 1-10)

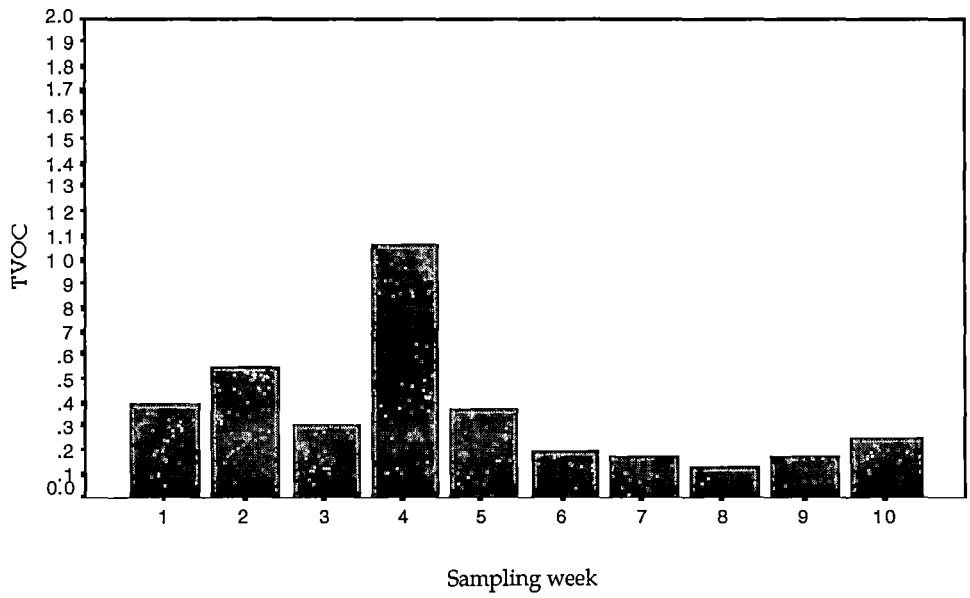


FIGURE 8.27

TVOC levels (mg/m^3) for building nine (weeks 1-10)

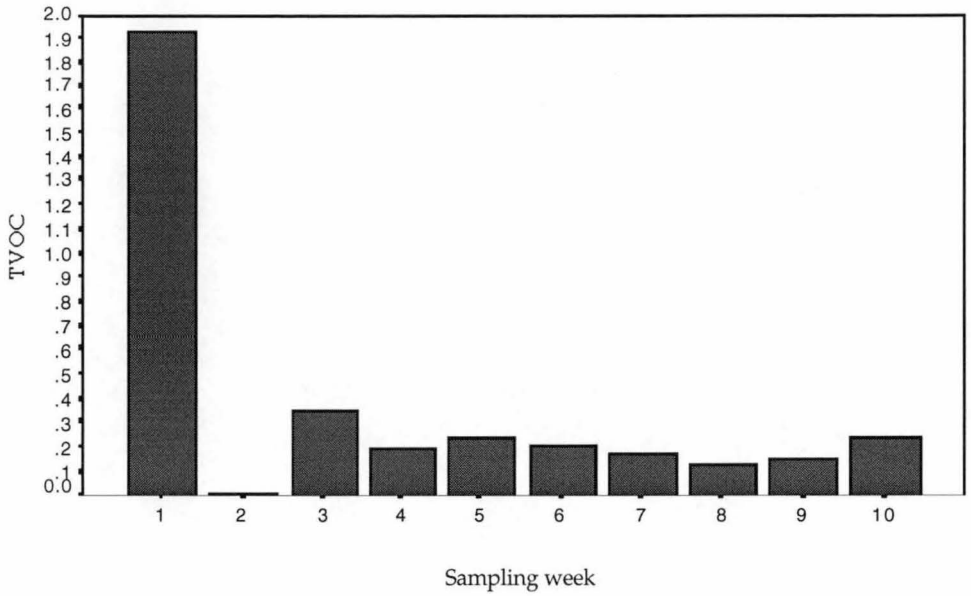


FIGURE 8.28

TVOC levels (mg/m^3) for building ten (weeks 1-10)

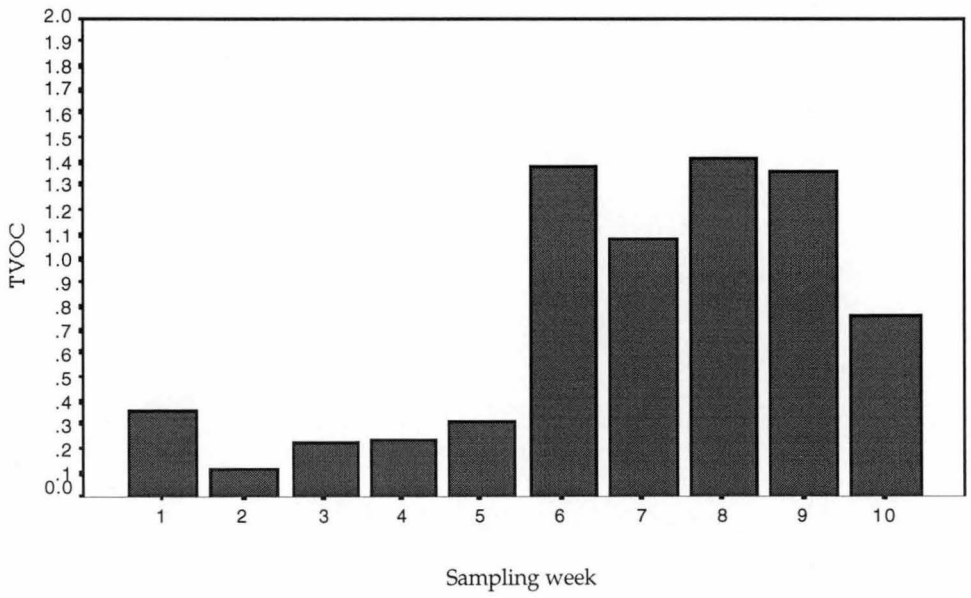


FIGURE 8.29

TVOC levels (mg/m^3) for building eleven (weeks 1-10)

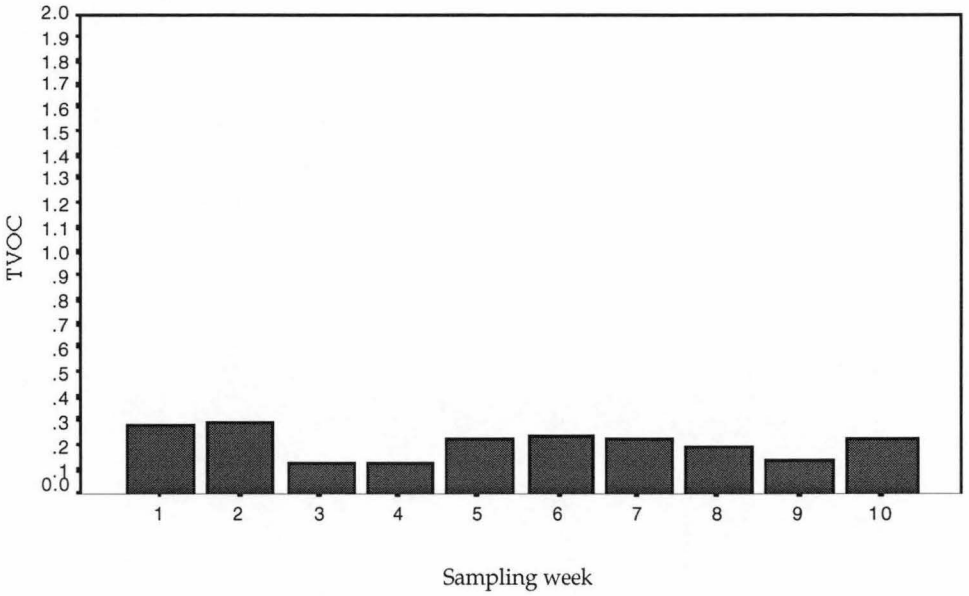
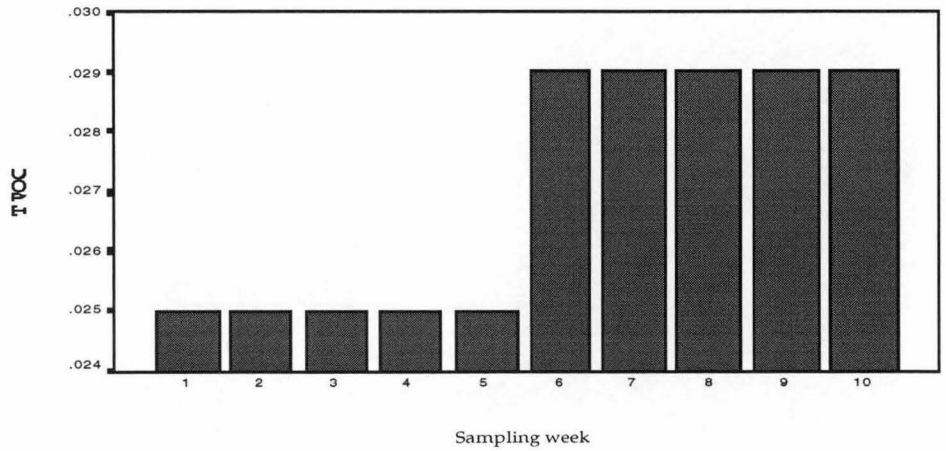


FIGURE 8.30

TVOC levels (mg/m^3) for the control building (weeks 1-10)
Note: the scale on this graph is different to graphs 8.19-8.29
to highlight TVOC levels



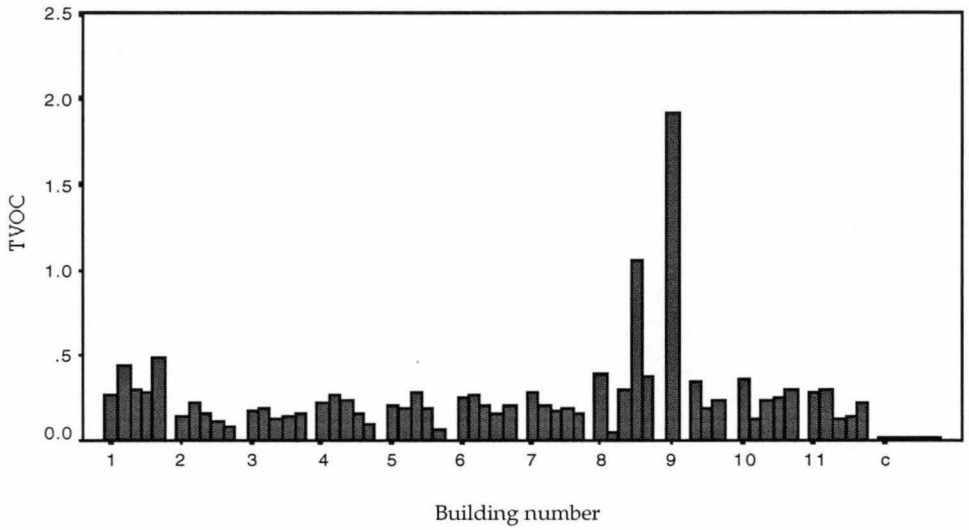
8.3.2 SEASONAL VARIATIONS IN CONCENTRATIONS

The seasonal variations are seen in figure 8.31, where winter TVOC levels (mean rank= 1.64) are consistently higher than in the summer months (mean rank= 1.36).

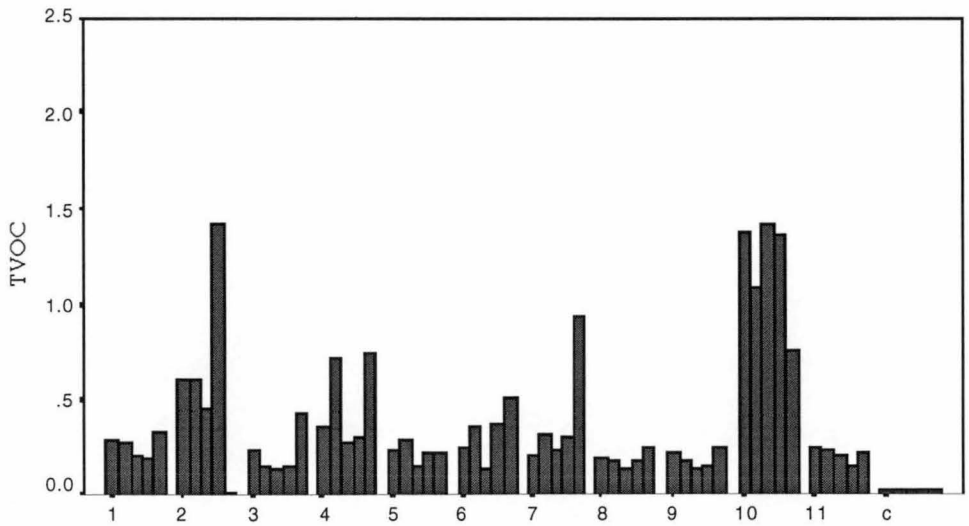
FIGURE 8.31

Winter and Summer (weeks 1-10) TVOC levels (mg/m^3) in sampled buildings (including control)

Summer (weeks 1-5)



Winter (weeks 6-10)



Results of the Friedman Two-Way ANOVA and the Wilcoxon Signed Rank Test indicate that significant differences did exist between the two seasons ($p<.05$). The exceptions to these are building 1 and building 8 where the high levels of isobutane and *n*-butane elevate summer TVOC levels to exceed winter values. Possible reasons for the seasonal TVOC variations include changing HVAC operations, occupant patterns, indoor/outdoor temperature differences and material emissions.

8.4 Volatile Organic Compounds and Other Variables

8.4.1 TEMPERATURE

During sampling in the summer months the indoor temperature in weeks 1-5 was more or less constant during the day (while the building was occupied) at 21.5 - 24.1 °C, but in winter it fluctuated between 18 - 25.2°C. Outdoor temperatures ranged from a minimum of 4 °C and a maximum of 25°C in the summer, and a minimum of 4.9 °C and a maximum of 20°C in the winter.

Examination of Spearman's Rank Order Correlation revealed that TVOC levels were not significantly correlated with indoor temperature (>.05), but were negatively significant with mean outdoor temperature (<.05) (figure 8.32), and positively significant with minimum outdoor temperature (<.05) (figure 8.33). These results, were also reflected in the Chi-square test for relatedness where mean outdoor temperature was related to TVOC with a significance value of p =.00000, which is well under the alpha level of .05.

Overall, weather conditions determine the amount of heating, cooling and humidity treatment required in a building. It has been outlined by Maroni *et al.* (1995) that temperature extremes limit ventilation based on the thermal conditioning capacity of the heating and cooling systems, giving the opportunity for contaminant concentrations to increase indoors. Therefore it is possible that the correlation between minimum and mean outdoor temperature and TVOCs was significant for the following reasons:

1. The outdoor air supply may have been decreased (due to changes in HVAC operations) therefore indoor air contaminants were allowed to build up rather than dilute. It has been claimed that increasing the volume of outdoor air supply tends to dilute indoor air contaminants;
2. The occupant patterns may have changed e.g. individuals wore more drycleaned clothes (1,1,1-trichloroethane) because of the colder weather; and

3. There may have been influences other than temperature such as wind speed and direction.

When values were transformed to a natural logarithmic transformation, a Bivariate Pearson Product-Moment Correlation indicated that both mean outdoor temperature (r = -.0945, p>.05), and minimum outdoor temperatures (r= -.0671, p>.05) had nonsignificant negative correlations with TVOCs, this is illustrated in figure 8.34.

Thus, increases in TVOC levels are more likely to be attributed to outdoor temperature or other variables, rather than indoor temperature fluctuations.

8.4.2 HUMIDITY

The indoor relative humidity was about 39% in the colder months, increasing to over 60% in the summer months. Outdoor humidity values ranged from 49-91% in the summer months to 53-91% in the winter.

As with temperature, Spearman's Rank Order Correlation revealed that there was no relationship between indoor relative humidity (>.05), outdoor relative humidity (>.05) and total volatile organic compound levels. These results were repeated when the Pearson Product-Moment Correlation was applied (r= -.0231, p>.05) to logarithmically transformed values.

FIGURE 8.32

Scattergram of non-logarithmically transformed TVOC levels and mean outdoor temperature

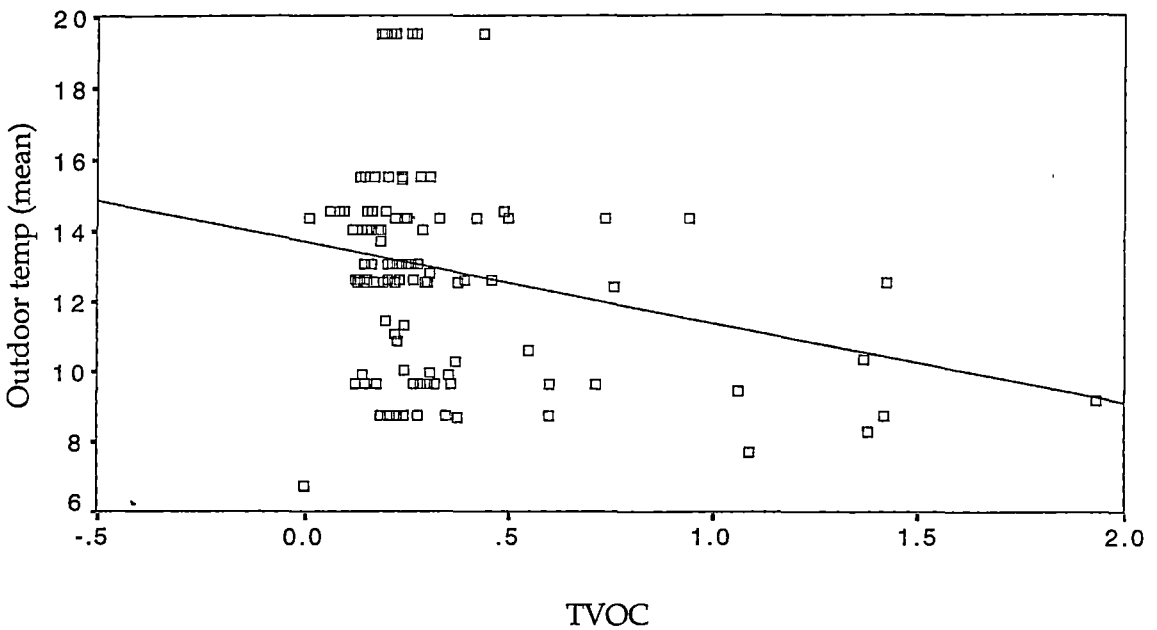


FIGURE 8.33

Scattergram of non-logarithmically transformed TVOC levels and minimum outdoor temperature

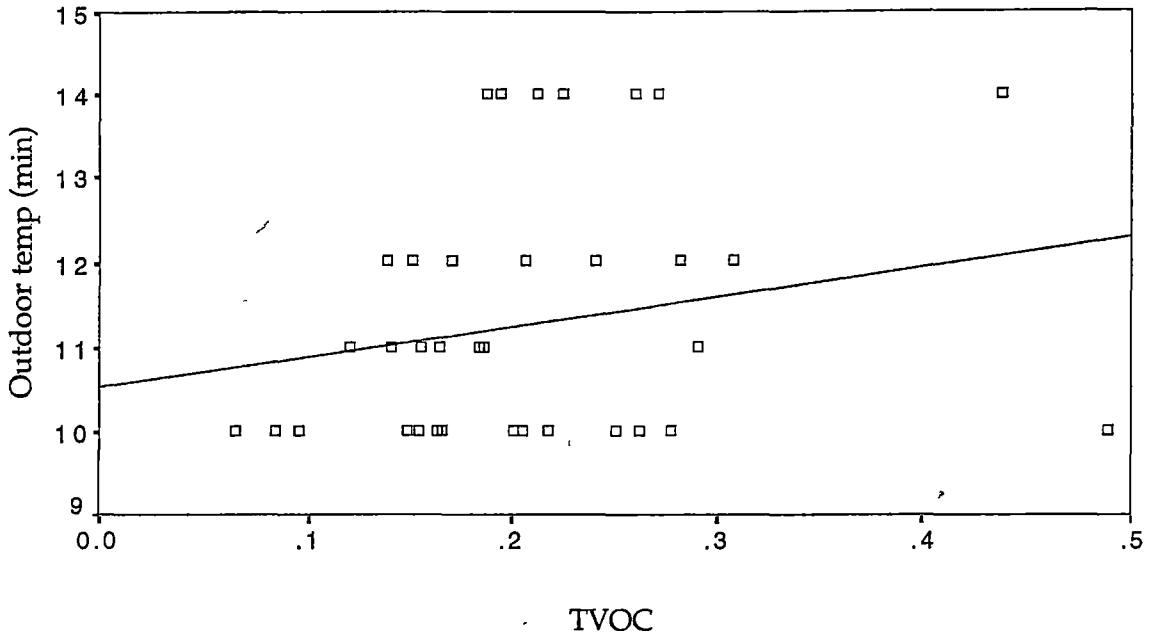
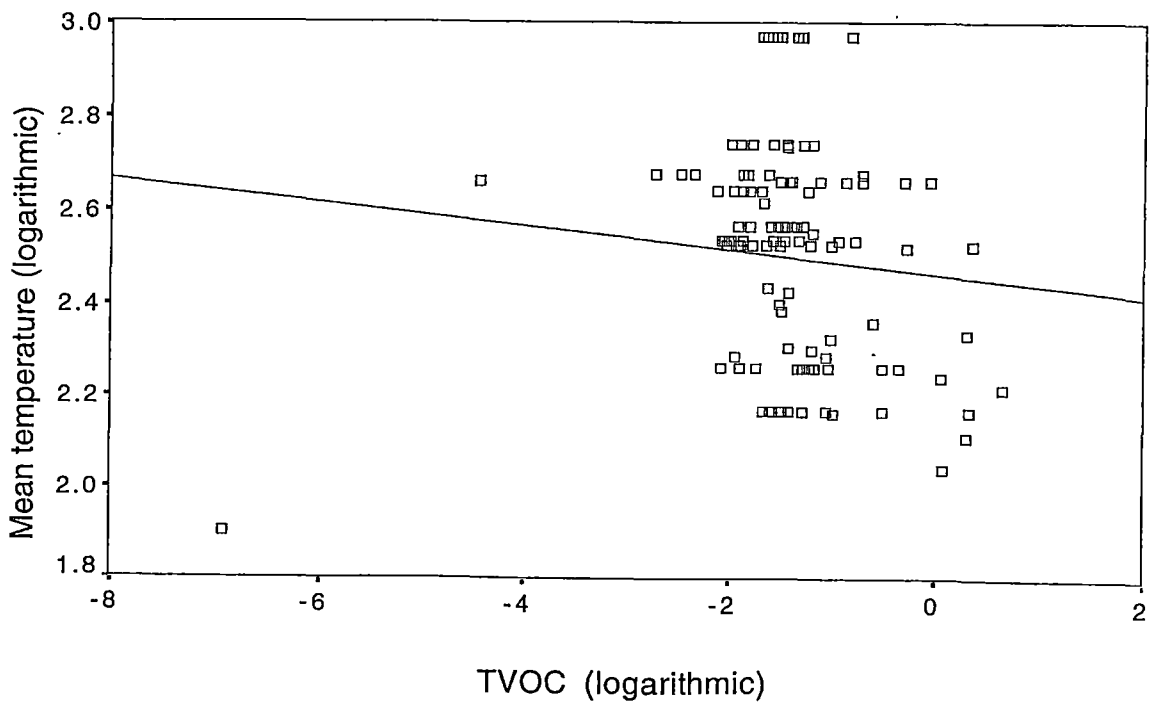


FIGURE 8.34

Scattergram of logarithmically transformed TVOC levels and mean outdoor temperature



8.4.3 BUILDING FACTORS

Each building factor (i.e. various building materials, room density, and outdoor air supply per person) recorded in the building information questionnaire was compared to the TVOC levels. The results from the Chi-square Test for Goodness of Fit indicated that none of the building factors had any significant effect with TVOC levels, as all variables were above the alpha level of .05.

8.5 Conclusion

The range and levels of hydrocarbons found in the sampled buildings were consitent with those found in other VOC studies. Winter TVOC values exceed summer values, but interestingly indoor temperature and humidity had no effect on organic compound concentrations. Overall, outdoor temperatures had more of an influence on TVOC levels than any other variable.

CHAPTER NINE

RESULTS PART TWO - FINDINGS of the QUALITATIVE ANALYSIS

9.1 Introduction

This chapter presents the results of the questionnaire survey of 265 workers (136 males and 129 females), following the methodology used for qualitative analysis outlined in chapter seven.

The ubiquity of VOCs and the casual approach to their use almost ensure contact with the lungs and the skin. Exposure to these low VOC concentrations, such as those identified in the quantitative analysis of this study, could possibly cause some general and specific effects on health.

The length of time in which employees in this study are exposed to VOCs is extensive. Overall, these exposure conditions generally last for long periods (7-8 hr per day × 5 days per week), and are, in the majority of cases, in areas where there are low levels of VOCs. This scenario is representative of the majority of settings in which we work, namely the office environment.

9.2 Individual Analysis of Symptoms

9.2.1 SICK BUILDING SYNDROME LEVELS

As seen in figures 9.1 and 9.2, 75% of buildings had more than 70% of employees reporting one or more symptom while in the work environment during weeks 1-5 of sampling. Similarly, 83% of buildings had >70% of employees reporting one or more symptom during weeks 6-10 of sampling. The control building on the other hand (weeks 1-10) had one building occupant out of three reporting symptoms, but attributed the symptom to a factor other than the office environment.

According to a working group of WHO (1982), an increased prevalence of nonspecific symptoms, occuring singly or in combination, in more than 20-25% of the workforce indicates a "sick building". When this conventional definition is applied to the study results, the majority of buildings fall into this category, and therefore can be defined as a "sick" (figure 9.1 and 9.2).

Once overall sickness levels have been quantified (appendix 7.1), it is necessary to examine in more detail the types of symptoms reported (established by a questionnaire) by building occupants in order to confirm a diagnosis of SBS. Generally, the symptomatology of SBS is varied, yet there are some distinguishing symptoms including nasal irritation, eye irritation, dryness of throat, irritation of the skin, fatigue, malaise, headache, and feelings of heavy headedness (Maroni *et al.*

FIGURE 9.1

Percentage of building occupants having one or more sick building syndrome symptoms while in the building during weeks 1-5 of sampling

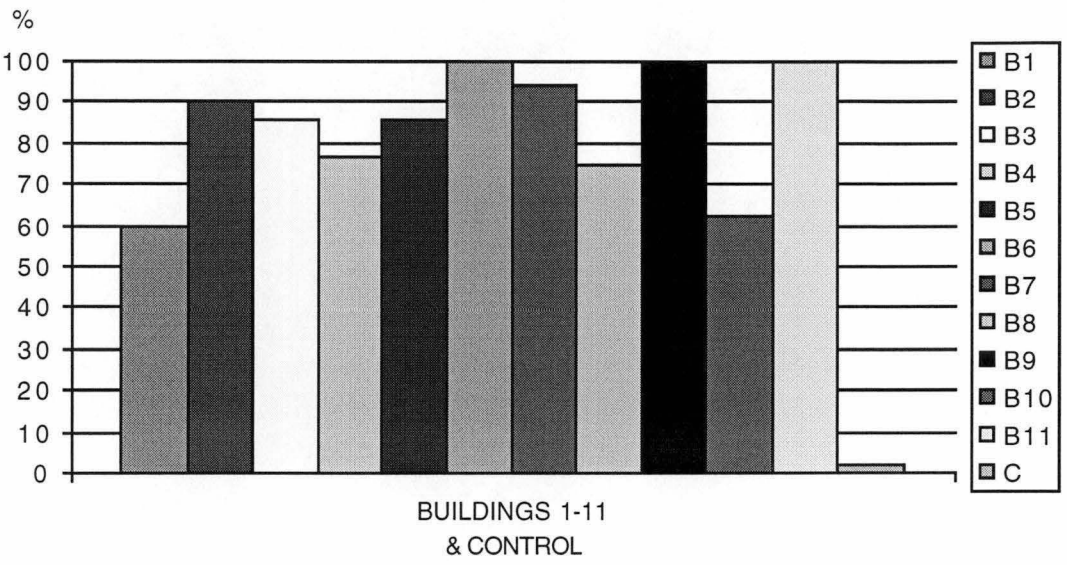
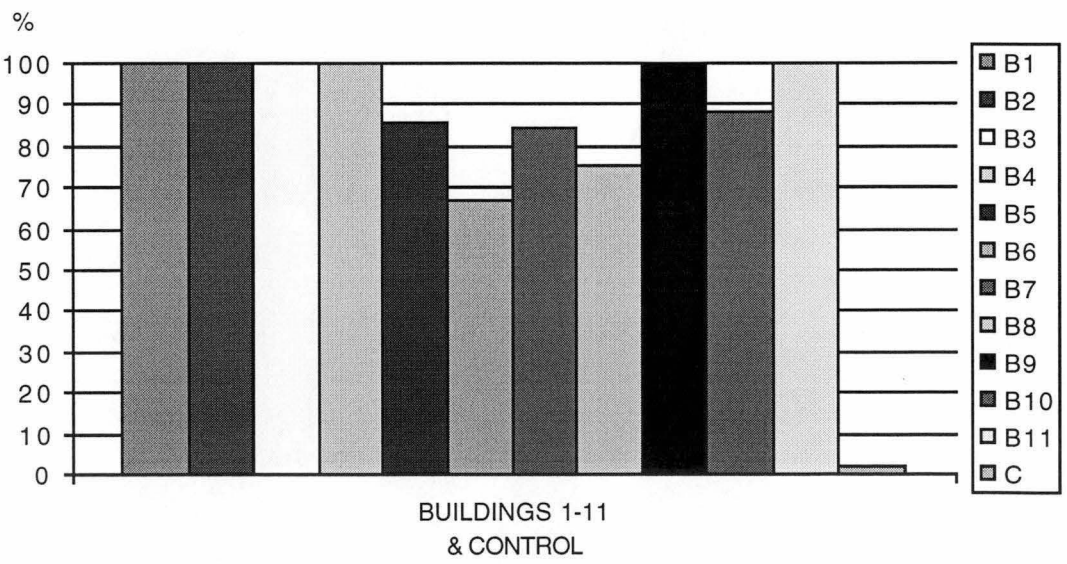


FIGURE 9.2

Percentage of building occupants having one or more sick building syndrome symptoms while in the building during weeks 6-10 of sampling



1995, WHO 1983). Therefore overall symptom levels were re-examined to only include three or more symptoms characteristic of SBS (including nasal, ocular, oropharyngeal, cutaneous and general manifestations categories).

These findings differ from the first analysis which summarised one or more possible symptoms (even if they were not typical of SBS). In the second analysis 50% of buildings had more than 70% of employees reporting three or more typical SBS symptoms while in the work environment during weeks 1-5 of sampling. Similarly, 54% of buildings had >70% of employees reporting three or more typical SBS symptoms during weeks 6-10 of sampling. This is illustrated in figures 9.3 and 9.4 which show the percentage of SBS symptoms attributed to the indoor environment in each of the sampled buildings (mean number of symptoms per individual=7.86).

It is clear that the extent to which individuals are reporting symptoms are in epidemic proportions in most surveyed buildings especially when compared to other Australian studies on office buildings (Kemp & Dingle 1994, Rowe & Wilke 1994) Although it is necessary to point out, that while these results are high, the nature and extent of symptoms could very well have been caused by other aetiological factors unrelated to indoor air exposure.

Another important point to elicit is the timing of the symptoms. It has been documented (Maroni *et al.* 1995, Robertson & Burge 1985, Valbjorn *et al.* 1990) that in practice, symptoms that are present on most days or in most weeks which improve on time away from the building, can be reasonably attributed to SBS. In order to examine this, the Spearman's Rank Order Correlation statistic was applied to the questionnaire survey results. Findings showed that reported symptoms in building occupants improve on days away from the building (<.05) and then recur on return (<.05).

9.2.2 CHARACTERISTIC SYMPTOMS

Table 9.1, illustrates the type of symptoms experienced while in the work environment. Twenty six different symptoms were reported, with the majority of those typical of SBS. The symptoms identified during the sampling period ranged from irritative symptoms of the eyes, nose and throat to cognitive, affective and cardiovascular effects. These symptoms are also consistent with other Australian studies on office buildings and homes (Dingle & Olden 1992, Rowe & Wilke 1994, Williams 1992).

In all cases the distribution of observed symptom levels were not normal. Each case was positively skewed and leptokurtic. The exceptions to this are the irritative symptoms of fatigue (-.591), headache (-.537) and sinusitis (-1.073) which had negative kurtosis values, indicating a flatter platykurtic distribution.

FIGURE 9.3

Percentage of building occupants having three or more sick building syndrome symptoms while in the building during weeks 1-5 of sampling

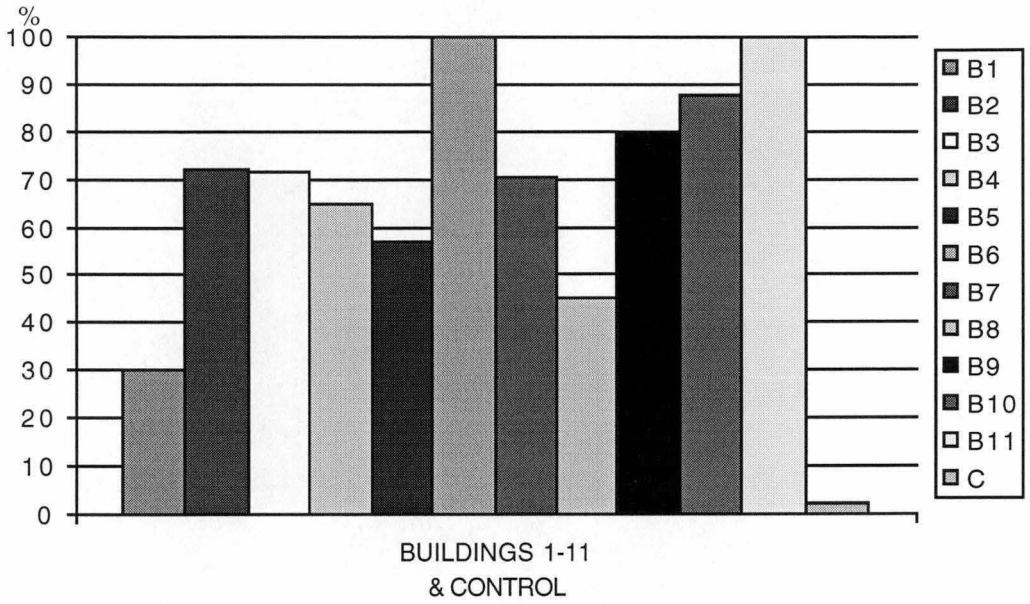
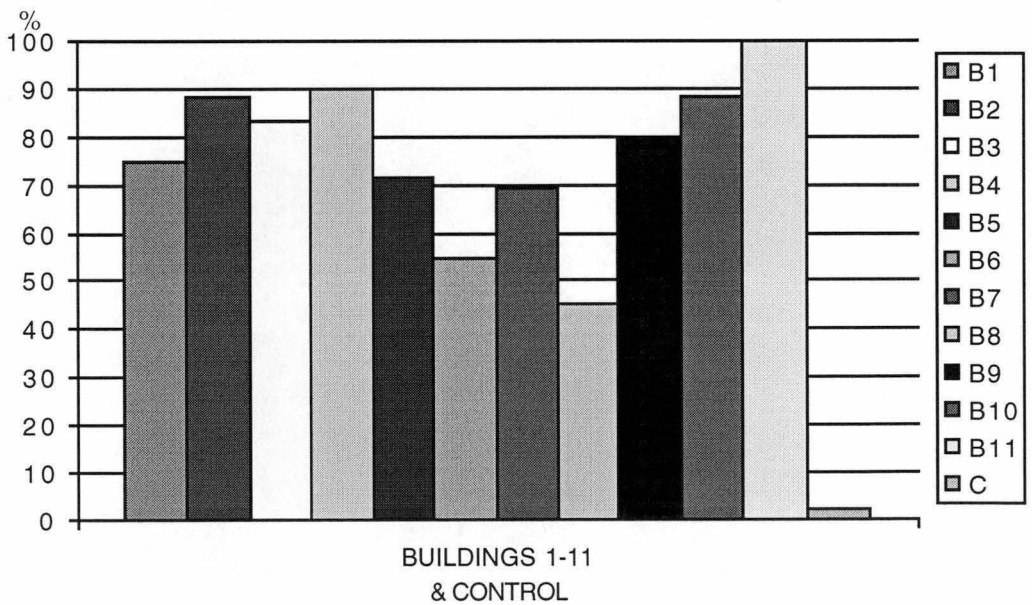


FIGURE 9.4

Percentage of building occupants having three or more sick building syndrome symptoms while in the building during weeks 6-10 of sampling



As seen in figure 9.5, fatigue was the most frequently reported symptom followed by stuffy/runny nose, headache, flu like symptoms, difficulties concentrating and eye irritation.

TABLE 9.1

Symptoms reported from a total sample of 265 individuals
(all buildings and the control) and their percentages

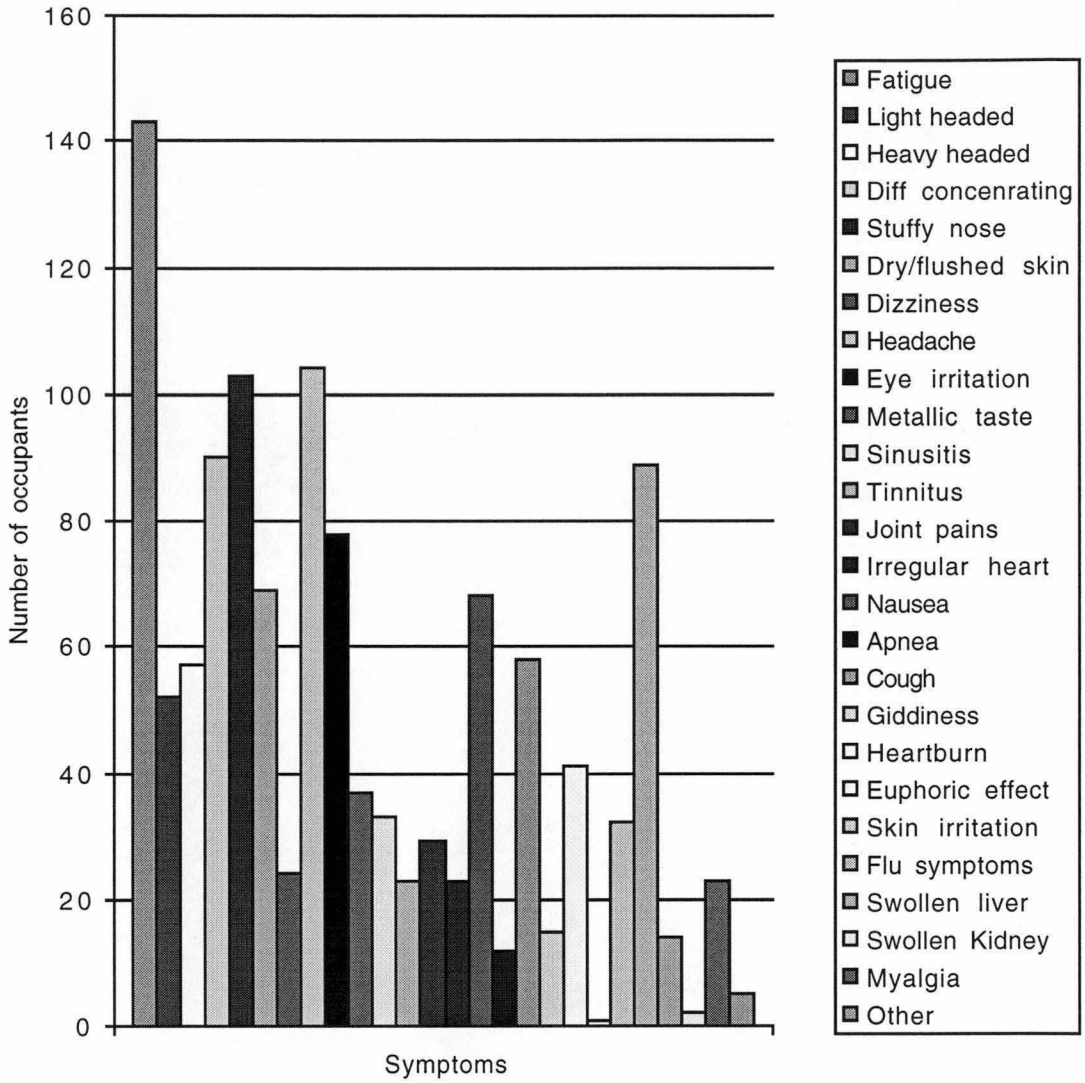
Symptom	Percentage	Symptom	Percentage
Fatigue	54%	Irregular heart rhythm	9%
Light headedness	20%	Nausea	26%
Heavy headedness	22%	Apnea	5%
Difficulties concentrating	34%	Cough	22%
Stuffy or runny nose	39%	Giddiness	6%
Dry/flushed facial skin	26%	Heartburn	15%
Dizziness	10%	Euphoric effects	2%
Headache	39%	Irritation of the skin on face	13%
Eye irritation	29%	Flu like symptoms	31%
Metallic taste	14%	Swelling of liver region	5%
Sinusitis	12%	Swelling of kidney region	2%
Tinnitus	9%	Myalgia	8%
Joint pains	11%	Other	2%

To determine whether reported SBS symptoms were associated with the indoor office environment, the Spearman's Rank Order Correlation was applied to survey results. Findings revealed that three characteristic SBS symptoms from the ocular category (eye irritation) and cutaneous category (dry and irritated skin), significantly improved after occupants left the workplace (<.05), and recurred when returning to the workplace (<.05). In addition, some symptoms (fatigue, stuffy or runny nose, giddiness and flu like symptoms), belonging to the general manifestations category, most noticably improved after leaving the work premises (<.001).

In addition, the Wilcoxon Signed-Rank Test indicated that all symptoms from the general manifestations category (i.e. fatigue, tiredness, and headache) were more often reported in the winter season (mean rank= 29.56) than in the summer (mean rank= 25.48) ($p<.001$).

FIGURE 9.5

Number of building occupants experiencing specific symptoms from a total sample (all buildings and the control) of 265 subjects



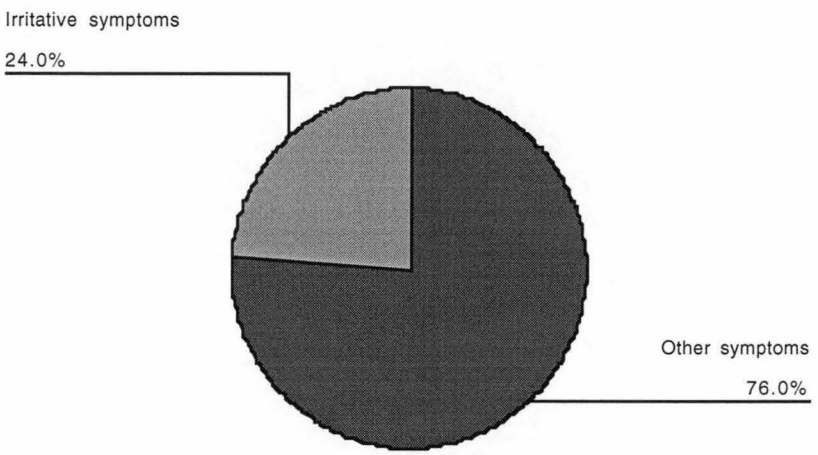
9.2.3 SENSORY IRRITATION

Figure 9.6 illustrates the percentage of irritative symptoms (stuffy/runny nose, dry or flushed facial skin, eye irritation, sinusitis, cough, and irritation of the skin) compared to other symptom types reported by the 265 subjects during the ten week sampling period. Overall, primary sensory irritation (direct stimulation of sensory cells by environmental changes) and secondary irritation (inflammatory changes in skin, mucous membranes and tissues) represented 24% of symptoms, compared to the remaining health complaints which make up 76% of the total symptoms.

Eye irritation and stuffy runny nose appear to be two of the most frequently reported, and the ones causing the greatest discomfort. Because these two anatomical sites are structurally related, it is quite likely that a strong relationship exists between the two.

FIGURE 9.6

Percentage of irritative symptoms compared to other reported symptoms



To examine this further the Bivariate Pearson Product-Moment Correlation was applied to logarithmically transformed irritative symptoms. Correlations appeared between eye irritation and stuffy nose ($r=.8492$, $p<.05$), and eye irritation and cough ($r=.7154$, $p<.05$). In addition, these irritative symptoms were reported more often in winter (mean rank= 113.26) than in summer (mean rank= 77.49). The Chi-Square Test confirmed this result ($p<.005$).

9.2.4 SYMPTOMS, PSYCHOSOCIAL FACTORS AND PAST DISEASE HISTORY

Generally, the number of symptoms was affected by conditions such as time of the day (mean rank=2.23), day of the week (mean rank=1.48), and season of the year (mean rank=2.30). The Friedman Two-Way ANOVA confirmed this result, where all three temporal conditions appeared to affect reported symptoms considerably ($p<.01$).

Further analysis using the Friedman Two-Way ANOVA revealed that some symptoms were more noticeable at certain times of the day (e.g. in the morning shortly after entering the workplace or in the late afternoon after a significant

amount of time has been spent in the office). Symptoms of heavy headedness, stuffy/runny nose, headache, eye irritation, cough and irritation of the skin were noted to be worse with different times of the day (<.05). Although at precisely what time of the day (morning/early afternoon/late afternoon) was not specified in the survey questionnaire. It was also noted that some symptoms (difficulties concentrating, dry/flushed facial skin, sinusitis, and joint pains) were noticeably worse on difference days of the week. Results were well below the alpha level of .05, but similar to the time of the day category, it was not possible to identify from the questionnaire which days of the week appeared worse/better.

Seasons of the year characterised a quite different range of symptoms when compared to the other two temporal conditions outlined. Symptoms such as fatigue ($p<.05$), lightheadedness ($p<.05$), giddiness ($p<.05$) and metallic taste in the mouth ($p<.05$) were reported by building occupants as varying with seasons of the year. Application of the Friedman Two-Way ANOVA test quantified this association.

Past disease history had noticable effects on the type of symptoms reported. Of all the past disease variables, allergic disorders (mean rank=3.36) had the largest influence, followed by hayfever (mean rank=2.73). The Friedman Two-Way ANOVA confirmed this result ($p<.05$).

To examine this further, each symptom, and their association with past disease history was analysed. Results revealed that symptoms most affected by pre-existing health conditions (allergies, hayfever and asthma) were fatigue ($p<.05$), difficulties concentrating ($p<.05$), sinnusitis ($p<.05$), stuffy/runny nose ($p<.05$), flu like symptoms ($p<.05$), irregular heart rate ($p<.05$) and giddiness ($p<.05$).

Overall, psychosocial factors (questionnaire numbers 15-18) had no effect on symptom reporting as alpha levels were greater than .05.

9.2.5 SYMPTOMS AND POPULATION VARIABLES - AGE, SEX, LENGTH OF TIME EMPLOYED AND EXTRANEOUS FACTORS

9.2.5.1 AGE

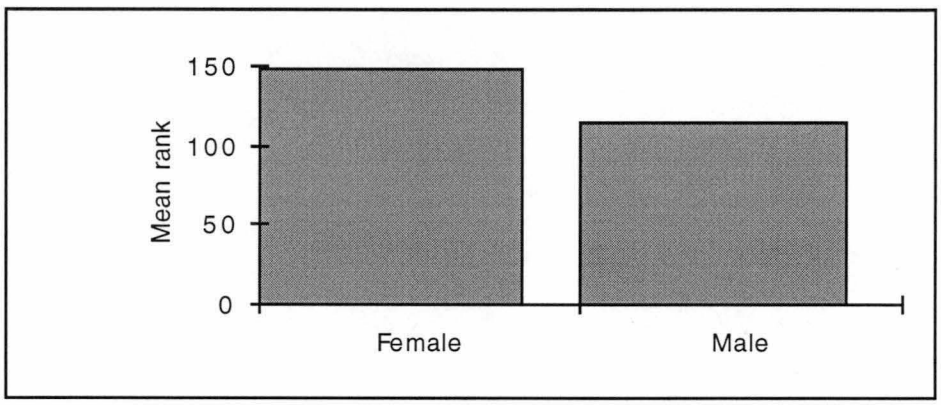
The Kruskal-Wallis One-Way ANOVA showed that the prevalence of symptoms reported varied across age groups (58 - 48 yrs, 47 - 38 yrs, 37 - 28 yrs, 27 - 18 yrs). Individuals between the ages of 38 - 47 yrs (mean rank=143.57) reported the most symptoms, followed by the age group 18 - 27 yrs (mean rank=129.62), although these were not statistically significant ($p>.05$). In addition, the age group of 38 - 47 yrs suffered from more hayfever ($p<.05$) and eczema ($p<.05$) than any other group in the study.

9.2.5.2 SEX

As seen in figure 9.7, results of the Mann-Whitney U test (Wilcoxon Rank Sum W Test), highlighted sex differences in symptom reporting. Overall, females (mean rank=149.05) significantly experienced more SBS symptoms while in the work environment than males (mean rank=115.34) ($p<.05$).

FIGURE 9.7

Sex differences in symptom reporting



9.2.5.3 LENGTH OF TIME EMPLOYED

The Kruskal-Wallis One-Way ANOVA showed that the year of employment influenced the number of symptoms reported. Four year groups (1961-1970, 1971-1980, 1981-1990, and 1991-1998) were examined and individuals who have been working in the buildings for between 18-27 years (mean rank=185.00) reported the most symptoms (<.05). It appears that the longer individuals are employed in a building, the more likely they are to develop health problems related to the indoor environment, although this is difficult to quantify because increases in symptoms maybe just a factor of age rather than any other variable.

9.2.5.4 TEMPERATURE AND HUMIDITY

It has been documented that temperature and humidity affects the nature and extent of symptoms reported in systematic building investigations. In order to examine this the Spearman's Rank Order correlation was applied to the reported symptoms. Results demonstrated that variables such as temperature (>.05) and humidity (>.05) effects were not associated with SBS symptoms.

9.3 Conclusion

The symptoms reported in this study are similar to those described in the literature, and highlight the extent to which individuals suffer from a variety of ailments while in the indoor work environment. Even though there are variations in signs and symptoms, patterns have emerged with the successive reporting of some symptoms over others. This is especially the case for typical SBS symptoms and sensory irritation. Cardiovascular and systemic effects have been noted a number of times, and although no firm evidence exists in the causal link between these effects and IAP, its frequency in reporting makes it noteworthy.

A few of the symptoms reported are similar in character to the effects of higher level solvent exposure (like giddiness), although a definitive toxicological relationship is not possible in this study, it appears that low level exposures may be having similar physiological effects to high vapour concentrations.

CHAPTER TEN

RESULTS PART THREE - QUANTITATIVE and QUALITATIVE ANALYSIS COMPARED and CONTRASTED

10.1 Introduction

Once the volatile organic compounds have been quantified and the symptomatology defined (chapters 8 and 9), the relationship between the two should be investigated. Therefore this chapter presents the combined results of the qualitative and quantitative investigation following the methodology outlined in chapter seven.

It is has been documented that exposure to individual VOCs cause acute and chronic long term health effects. For example, common low grade symptoms such as skin and eye irritation can be suggestive of exposure to ethanol. But, of more recent and equal concern, are the exposures to truly complex mixtures of VOCs (such as the TVOCs identified in this study). To evaluate the relationship between individual VOCs/TVOCs and symptomatology further, each chemical element is examined in isolation, as well as its combinations, to determine possible links to illhealth in the non-industrial environment.

10.2 Relationships Between Individual Volatile Organic Compounds and Symptoms

The effects seen in organic solvent exposure will depend on many factors including solvent composition, exposure level, frequency and coexposure, and subject sensitvity. As seen in table 10.1, Spearman's Rank Order Correlation Coefficient results reveal that individual volatile organic compounds pertaining to a specific molecular class have a stronger association with certain symptoms.

Closer examination of the data (table 10.1) reveals that 1,1,1-trichloroethane (a chlorinated hydrocarbon) and ethanol (an alcohol) had more correlations with symptoms than any of the other compounds. When reviewing only irritative symptoms (stuffy/runny nose, dry or flushed facial skin, eye irritation, sinusitis, cough and irriation of the skin), terpenes had the most significant effect on sensory irritation followed by the aromatic, aliphatic and alcohol families.

When observing the effects on target organs, results of the sampled building population are almost identical to findings highlighted in toxicological tests (appendix 3). For example, elevated levels of 1,1,1-trichloroethane in this study were correlated with increases in irritation of the eyes and skin. These are the same target organs associated with both acute and chronic exposure to this compound (Sigma-Aldrich 1996). Similarly toluene exposure appeared to affect the liver, kidney and brain, and *n*-butane the eyes causing irritation and flu symptoms, but this was not confirmed by any pathological tests e.g. liver function tests.

TABLE 10.1

Summary of individual VOCs and their correlations with symptoms
(significance level <.05)

	Volatile organic compound														
Symptoms	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
A									♦			♦			
B				♦	♦	♦	♦		♦						
C							♦		♦		♦				
D									♦			♦			
E	♦							♦	♦			♦			
F							♦		♦			♦			
G				♦			♦								
H									♦			♦			
I		♦						♦	♦	♦	♦	♦			
J						♦									
K			♦						♦		♦	♦			
M						♦			♦						
N						♦									
O	♦	♦		♦							♦				
S			♦	♦		♦		♦	♦	♦		♦			
T					♦			♦				♦			
U		♦					♦	♦				♦			
V	♦			♦	♦	♦		♦		♦	♦				
W					♦										
X					♦	♦									
Y	♦			♦	♦				♦			♦			

LEGEND

Chemical name - 1 isobutane, 2 n-butane, 3 acetone, 4 benzene, 5 toluene, 6 ethylbenzene, 7 xylenes, 8 1,2,4-trimethylbenzene, 9 1,1,1-trichloroethane, 10 α-pinene, 11 limonene, 12 ethanol, 13 n-octane, 14 n-nonane, 15 n-decane.

Symptom - A fatigue, B light headed, C heavy headed, D difficulties concentrating, E stuffy or runny nose, F dry/flushed facial skin, G dizziness, H headache, I eye irritation, J metallic taste, K sinusitis, N irregular heart rhythym, O nausea, S heartburn, T euphoric effects, U irritation of facial skin, V flu like symptoms, W swelling of liver, X swelling of kidney, Y myalgia.

Once again, these are reflected in toxicological tests (to lower level compounds) which highlight the same outcomes. It has been demonstrated by the results that we may have been underestimating the health effects of individual compounds that are well below toxic levels or exposures to complex mixtures.

10.3 Relationships Between Total Volatile Organic Compounds and Symptoms

10.3.1 SBS SYMPTOMS AND VOC MIXTURES (TVOC)

The Bivariate Pearson Correlation Moment Correlation highlighted a significant positive relationship between three or more reported SBS symptoms (including sensory irritation) in all sampled buildings (excluding the control building), and TVOC levels which have been logarithmically transformed (r=.1789, <.05). Thus, increases in symptoms are positively associated with elevated TVOC values, or mixtures of compounds, but the relationship is basically non linear and very weak (r=.031). This is illustrated in figure 10.1, which shows a scattergram of this result, its regression line, mean predicted response (95% confidence interval), and single observed response (95% confidence interval). In contrast, remaining symptoms reported (those not considered characteristic of SBS), were not correlated with TVOC levels (r=.1548, >.05).

Sixty four percent of sampled buildings (including the control building) had TVOC concentrations within the TVOC no effects range of <.20 mg/m^3 (comfort range). The remaining 36.4% of buildings are well above the .20 mg/m^3 level, or range suspected in causing discomfort and irritation in building occupants (multifactorial exposure range).

It can be concluded from the results that a reasonable proportion of TVOC concentrations are in the no effects range. In spite of this, characteristic SBS symptoms (excluding skin and mucous membrane irritation) (41%) and irritative symptoms (40%) still affected the building population within this TVOC category as seen in figure 10.2 and figure 10.3. In contrast, 60% of reported irritative symptoms and 59% of characteristic SBS symptoms (excluding skin and mucous membrane irritation) were in the TVOC range of 0.20-3.0 mg/m^3.

It appears that both SBS symptoms (excluding skin and mucous membrane irritation) and irritative symptoms are more frequent in the multifactorial range. Results revealed that TVOCs in the multifactorial range had 20% more reported symptoms (SBS and irritative) when compared to the no effects range.

FIGURE 10.1

Scattergram displaying the correlation between logarithmically transformed TVOC levels and SBS symptoms.

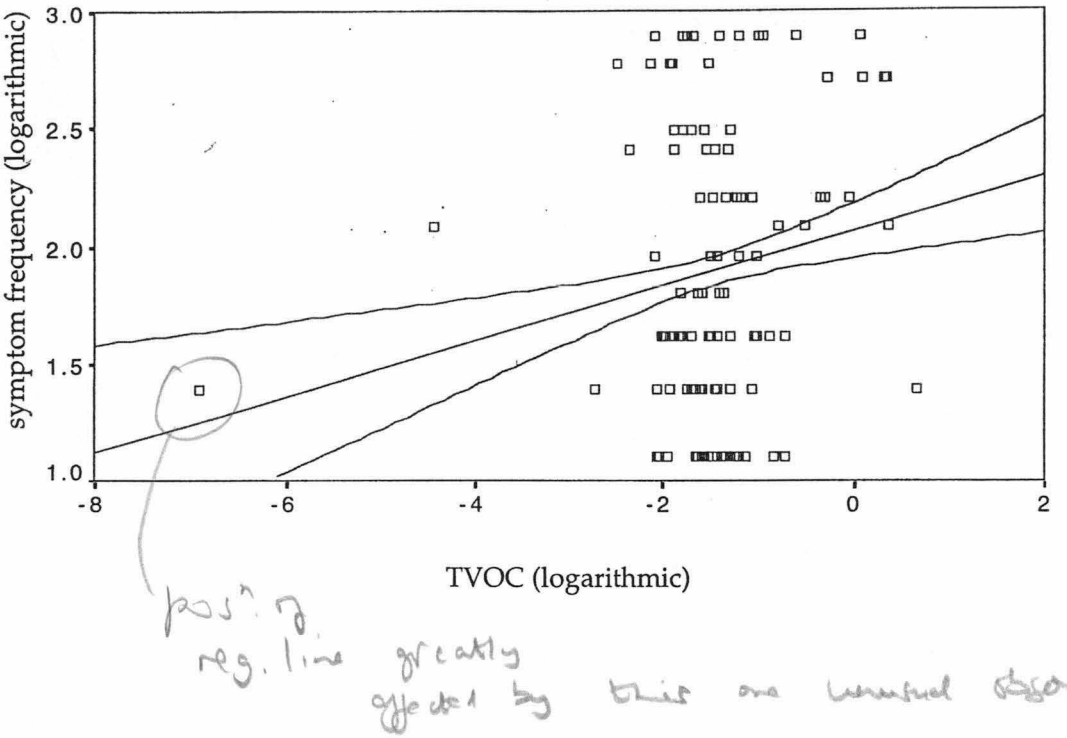


FIGURE 10.2

Reported SBS symptoms (excluding skin and mucous membrane irritation) and their percentages in the TVOC "no effects" and "multifactorial" range

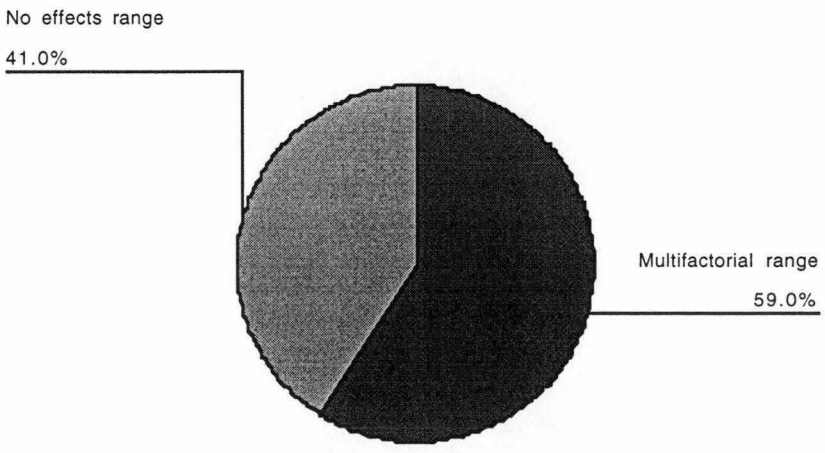
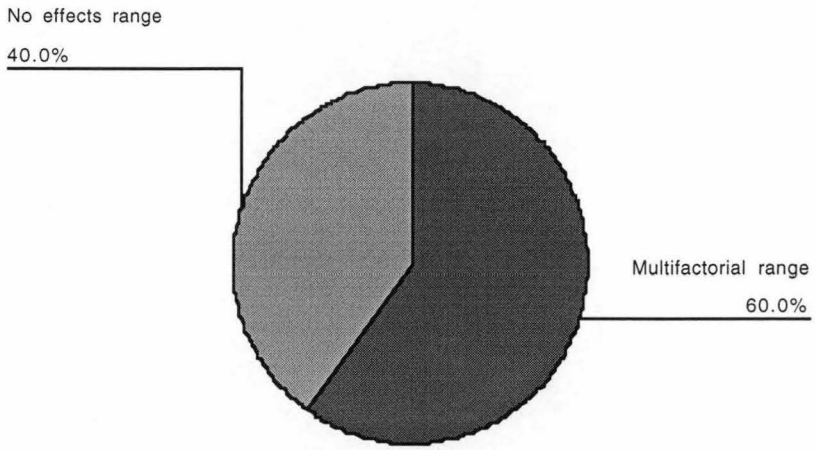


FIGURE 10.3

Reported irritative symptoms and their percentages in the TVOC "no effects" and "multifactorial" range



It has been documented (Molhave 1990), that in the range 0.20-3.0 mg/m^3, health effects and discomfort would be expected to occur if other factors interact with TVOC exposures such as temperature and humidity. The TVOCs' reasonable interaction with mean outdoor temperature in this study has been highlighted earlier, and would appear to fit within this category.

10.3.2 VOC MIXTURES AND INDIVIDUAL SYMPTOMS

The Bivariate Pearson Product-Moment Correlation was applied to logarithmically transformed symptoms in conditions of lower TVOC values (<.02 mg/m^3). It was concluded that a significant positive relationship exists between fatigue and light headedness ($r=.6773$, $p<.05$), heavy headedness and difficulties concentrating ($r=.8266$, $p<.05$), difficulties concentrating and dry flushed facial skin ($r=.5460$, $p<.05$), and irregular heart rhythm and nausea ($r=.7148$, $p<.05$) in the presence of lower TVOC levels. Heavy headedness and difficulty concentrating were also significantly correlated in the Spearman's Rank Order Correlation (<.05).

In most cases, a significant positive relationship is evident between fatigue and light headedness ($r=.7148$, $p<.05$), heavy headedness and difficulties concentrating ($r=.8266$, $p<.05$), and irregular heart rhythm and nausea ($r=.7148$, $p<.05$) in conditions of higher TVOC levels (0.2-3.0 mg/m^3). This was confirmed in the Spearman's Rank Order Correlation where increased reporting of difficulties concentrating (<.05) was associated with TVOC levels in this range.

Interestingly, the "no effect" level in this study does cause both irritation and discomfort, but not to the same extent as the multifactorial exposure range. Other systematic building studies have highlighted similar results where SBS symptom prevalence rates and sensory irritation have been evident in conditions where TVOC levels are in thresholds lower than 0.20 mg/m^3 (Norback *et al.* 1990b). It is likely that additional factors such as other indoor pollutants influenced the higher incidence of symptoms in the "no-effects range". Indoor contaminants that were not measured in this study such as MVOCs, physical pollutants, and other organic compounds may have caused symptoms highlighted in this study. Irrespective of these additional factors SBS symptoms were still higher in the range above 0.02 mg/m^3 therefore it can be concluded that VOCs and TVOCs can contribute and influence increases in symptoms characteristic of SBS.

10.4 Further Examination of the TVOC

As seen in table 10.2, aliphatic hydrocarbons (Alkanes), aromatic hydrocarbons, and alcohols and ketones, appeared to have a strong influence on TVOC levels. Spearman's Rank Order Coefficient revealed that specific species of VOCs were associated with higher overall TVOC levels (<.05).

TABLE 10.2

Volatile organic compounds that have the strongest association with TVOC values

Volatile Organic Compound	Spearman Correlation
Ethanol (Oxygenated hydrocarbon)	.00000
Acetone (Oxygenated hydrocarbon)	.00000
n-butane (Aliphatic hydrocarbon)	.00000
Isobutane (Aliphatic hydrocarbon)	.00000
1,2,4 -trimethylbenzene (Aromatic hydrocarbon)	.00000
Limonene (Aromatic hydrocarbon)	.00000
Toluene (Aromatic hydrocarbon)	.00001
Xylenes (Aromatic hydrocarbon)	.00002

The presence of these eight chemicals appears to be critical in distinguishing a "sick" and a "healthy" building. Sampled buildings, defined as "sick", all contained each of these eight key chemicals, whereas the control building,

categorised as "healthier", had traces of only three of these critical substances (acetone, toluene and *m-*, *p-*, *o-*, xylenes).

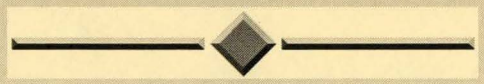
By examining this result it seems appropriate to suggest that when given a certain combination or quantity of chemical subtances in a building, one could reasonably expect a proliferation of sickness levels, no matter what their clinical manifestation. In contrast, the absence of one or more compounds may reduce the overall amount of chemical transformations, potentially reducing the exposure to oxidative or reactive species believed to cause irritation.

Therefore the presence or absence of certain chemical compounds, such as the one highlighted by this result, could possibly be a key factor in determining why some building occupants have higher observable symptoms (i.e. SBS) than others.

10.5 Conclusion

It appears from the results that both VOCs and TVOCs have effects on the health of individuals in the non-industrial environment. TVOCs in the multifactorial exposure range have more of a significant influence on sickness levels than those in the comfort range. Interestingly, reporting of symptoms was still quite high in what is regarded as the comfort or "no effect" range. The type and quantity of VOCs present in a building appear to influence sickness levels, with some key organic compounds suggested as conducive to development of SBS. But it must be taken into account that other indoor air variables including lighting, noise, electromagnetic fields and particles were not measured but would have undoubtedly influenced results.

CHAPTER ELEVEN



DISCUSSION

11.1 Introduction

Results of the quantitative and qualitative analysis in this sytematic building investigation revealed some findings that complement current VOC research. In contrast, some of the results, such as those associated with the TVOC concept, challenge conventional theories on the health effects of organic pollutants. This chapter therefore reviews each of the objectives in order to evaluate the underlying hypotheses presented in this building analysis, and examines and discusses further the outcomes and limitations of the study. To support the findings of this Tasmanian study, the discussion throughout this chapter continuously refers to other studies and information found in the indoor air quality literature for comparative purposes.

11.2 Discussion of Aims

11.2.1 DISCUSSION OF AIM ONE - *To assess the types and concentrations of hydrocarbons found in the indoor office environment*

Saturated open-chain hydrocarbons (alkanes), unsaturated cyclic hydrocarbons (aromatics), and oxygenated hydrocarbons (alcohols and ketones), were the most frequently detected. As seen in Table 11.1, these findings complement VOC field studies undertaken elsewhere. Aromatic and aliphatic compounds dominated all other VOC types. Of the aromatic class, benzene, toluene and xylenes were very common, which is not suprising because they are emitted from a large variety of indoor sources including building materials and photocopying machines (xylenes) (Leovic *et al.* 1996). In contrast, terpene concentrations (α-pinene and limonene) were consistently amongst the lowest levels detected. Given that terpenes based products (e.g. cleaning fluids) are used ubiquitously in consumer and commercial products, its presence at higher levels would have been anticipated.

Two points of interest are highlighted in the results. Firstly, ethanol was not detected in the majority of buildings during the summer months. As one might expect ethanol would be present continuously (although at varying levels), given its multitude of sources (solvents, fuel, biological origin). Other studies, such as the one undertaken by Santos *et al.* (1997), highlight the unexpected presence of ethanol in high levels during both the summer and winter months.

In this Tasmanian study, *n*-octane, *n*-nonane and *n*-decane were present only in buildings that had been contaminated with sovents or fuel components, but

without the presence of *n*-hexane, *n*-heptane, and other members of the alkane family. These two additional compounds should have been detectable in the Tasmanian study samples because all sites were located near vehicular emission sources, but the reason for their absence has not been determined.

TABLE 11.1

Compounds (mg/m^3) most frequently observed in 10 buildings (83 offices) of the European Parliament in Brussels, Luxembourg and Strasbourg
(Source: Maroni *et al.* 1995, p54)

Compound	Median (50% ile)	90% ile
1,1,1-trichloroethane	<.02	.040
xylenes	.005	.010
toluene	.011	.036
benzene	<.001	.0068
n-hexane	.002	.021
n-heptane	.001	.024
tetrachloroethene	.002	.16
formaldehyde	.045	.12
acetaldehyde	.009	.024

Compounds (mg/m^3) most frequently observed in 11 buildings taken from the Tasmanain VOC study

Compound	Median (50% ile)	90% ile
1,1,1-trichloroethane	.024	.088
xylenes	.018	.031
toluene	.025	.045
n-decane	.034	.135
n-butane	.032	0.90
n-nonane	.021	.100
isobutane	.018	.060
ethanol	.081	.344
ethylbenzene	.004	.018

According to Drahonovská and Gajdos (1997), the location of a building is more important than any of the materials used in its construction and furnishing. Therefore, if buildings are situated in the central business district, one would expect more alkanes to be present. In the reviewed literature, these related compounds have, in the majority of cases, been detected collectively in home and office air

samples. This view is consistent with the findings of this study because the highest concentrations of VOCs belonged to the alkane family, and all buildings were in locations subject to areas of high vehicular traffic, so there may have been an association between these two variables. It would therefore be reasonable to assume that contaminants from car exhaust played a large part in the presence of alkanes in the results, but this would have to be quantified. Or, the rise in alkanes may have been caused by a combination of factors such as general topography, contaminants generated on adjacent sites, prevailing wind patterns and wind velocities, management of the air system, or the configuration of neighbouring buildings which affect the dilution or escape of contaminant plumes that influence increases in VOC concentrations.

In this study, specific sources have been identified for the unsaturated cyclic hyrocarbons α-pinene and limonene. These are derived from predominantly consumer and personal products, especially detergents and other perfumed products which have large quantities of limonene. Further examination of the consumer and commercial products was undertaken by obtaining an ingredient list from the manufacturers, and the information supplied confirmed the presence of these chemicals in the products used in the sampled buildings. 1,1,1-trichloroethane's sources are numerous, and it was found at all sites in the study. The most obvious source of this compound is "white out" correction fluid, consumer and commercial products (i.e. waxes, window cleaners, room deoderants). Once again this was confirmed by information provided by the product manufacturers.

When examining the TVOC concentrations, some individual compounds appeared to be critical in differentiating bewteen a "sick" and "healthy" building. *n*-butane, limonene, isobutane, ethanol, acetone and 1,2,4-trimethylbenzene were all at significant levels in the buildings with reported high SBS prevalence rates. On the other hand the control building had only three of these same compounds (acetone, toluene and xylenes) at detectable concentrations. These results are similar to the findings of Baird *et al.* (1987), who were able to distinguish between sick and healthy buildings by the presence of several critical chemicals. They identified six of these including tetrachloroethene, α-pinene, 1,1,1-trichloroethane, butylacetate, *n*-dodecane and *n*-tridecane. In addition, it may be just the increased variety of chemical species that determine whether buildings will have the potential to cause problems in individuals, rather than the type of chemicals present.

Other factors can also play an important part in the detection and distribution of VOCs. Ventilation, adsorption (Vanderwal *et al.* 1998), and other sink mechanisms which are not fully understood, all interact to determine the final concentrations of chemical compounds in the air. These factors may have influenced the outcome of detected VOCs in this study, but at this stage there are limited methods available to quantify this. In addition, the VOCs found in the

buildings may have, in some instances, been caused by a factor other than outgassing of materials and cleaning regimes. It is quite possible that some of the VOCs present (such as acetone), were due to microbial production of MVOCs in the HVAC system (Schleibinger *et al.* 1997, Wessen & Schoeps 1996).

11.2.2 DISCUSSION OF AIM TWO - *To evaluate if the types and concentrations of hydrocarbons vary on a weekly or seasonal basis*

The types and concentrations of hydrocarbons varied on a weekly basis in this study, illustrating the dynamic and diverse nature of the indoor environment. The strength of the emissions can be influenced by many variables, and isolating sources can prove difficult. Yet there are instances which are more clearly defined. The use of cleaning agents, for example, can result in a noticeable increase in the overall VOC levels (Wolkoff *et al.* 1998). This was highlighted in building BI, where a considerable rise (0.29 mg/m^3 in week 4) in TVOC levels was recorded following a carpet steam cleaning procedure (0.49 mg/m^3 in week 5). On the other hand other factors may have influenced results because there was a similar increase in the TVOC concentrations in building B1 between week 1 (0.26 mg/m^3) and week 2 (0.43 mg/m^3) (see table 8.2).

Seasonal variations were quite marked in this study, with winter values significantly exceeding summer concentrations (with the exception of two buildings). These results are identical to those found by Simoni *et al.* (1998), who noted that monitored pollutants in summer were lower when compared to winter levels. Crump *et al.* (1997) also found a significant rise in TVOC values during the winter in occupied homes, which was in contrast to unoccupied homes (lower TVOC values). This suggests that the winter peak in homes is due to occupant activities affecting source strengths and perhaps ventilation rate rather than any influence that outdoor temperature has on emission values from materials and furnishings. The two buildings that had elevated summer VOC concentrations may have been strongly influenced by a sporadic or episodic event, such as carpet cleaning, rather than any seasonal effects, but this has not been established in this study.

Temperature did have a noticeable effect on VOCs, but not indoor temperature fluctuations as expected. When reviewing the results of this study, it appears that colder outdoor temperatures have more of an association with VOCs than originally anticipated. Even though the correlation was weak, it is possible that the correlation between minimum and mean outdoor temperature and TVOCs was significant because of a variety of reasons including changes in HVAC operations (due to the colder weather), where decreased outdoor air supply subsequently allowed indoor air contaminants to build up rather than dilute. The differences in temperature may have changed occupant patterns, leading to an

increase in VOC concentrations (e.g. more drycleaned clothes were worn in winter). In addition there were other factors that may have influenced results such as wind speed and direction, but these were not measured in the study.

It was expected that the colder temperatures outdoors would promote the use of heating in a building, leading to an increase in indoor temperature, and a greater outgassing of VOC sources which subsequently elevate VOC concentrations (Crump *et al.* 1997). The results of this study fail to support this theory because fluctuations in indoor temperature had no correlation with VOC concentrations. It would therefore be reasonable to assume that the findings appear to be related to the minimalisation of fresh air due to colder outdoor temperatures (as outlined above), rather than indoor temperature variations.

The levels of humidity recorded in this study also had no correlation with TVOCs. It has been well documented that humidity has a similar effect to temperature (Smedje *et al.* 1997). Generally speaking, the higher the humidity values, the higher the VOC levels. Normally few problems occur when the relative humidity of indoor air is between 30-70% (assuming that no condensation occurs). In this study the indoor humidity was on average 39% in the colder months and 60% in the summer months, therefore the humidity values were within the range believed (Bienfait *et al.* 1991) to be acceptable and desirable. Any humidity value higher than 65% will enhance the emission of materials e.g. formaldehyde.

No building factors (HVAC operations, presence of carpet, basic design features) in the findings of this research significantly influenced VOC concentrations. These results complement other research findings in which building factors showed a nonsignificant correlation with variables such as ventilation system, carpets, heating, and symptoms (Gustafsson *et al.* 1996). It is difficult to determine this in the Tasmanian study because all sampled buildings had the same or very similar building elements (such as operation and maintenance of the building, occupants of the building and their activities, building contents, outdoor environment, building structure). If buildings with differing elements were sampled for VOCs then it would have been possible to more accurately compare and contrast whether VOC concentrations differed across building types.

11.2.3 DISCUSSION OF AIM THREE - *To determine and verify chronic low level human exposure to volatile organic compounds, particularly those of the aromatic and aliphatic class, and their subsequent health effects, while in the indoor environment*

11.2.3.1 GENERAL HEALTH EFFECTS FROM THE OFFICE ENVIRONMENT

All sampled buildings (except the control) have been defined as "sick" given the high incidence of work related symptoms, and confirm the findings of the 1995

Hobart study (Mesaros 1995). It has been claimed that occupants in a sick building report more ocular symptoms, upper airway disturbances and general symptoms (Bourbeau *et al.* 1997, Kroling 1998, Muzi *et al.* 1998) when compared to adequately maintained indoor environments. These findings are reflected in this study because no work related symptoms were reported in the control building (identified as having proper indoor air management strategies), in contrast to high symptom prevalences in the "sick" buildings. Whether the incidence of illness and clinical symptoms reported in this study were due only to the VOC concentrations in the indoor environment is unlikely. VOCs quantified in this study could have influenced some of the symptom rates and types, but the complexity of establishing a definite cause-effect relationship between the two is beyond the scope of this thesis.

It has been brought to attention in many publications that SBS can have, and in some cases does have, major causes unrelated to the building. For example, there is a school of thought that work related stress is an aetiology of sick building syndrome (Ooi & Goh 1997), particularly high levels of mental and physical stress (Bullinger *et al.* 1998). The results of this study provided an alternative view because psychosocial factors had no statistically significant effect on symptom reporting. That is not to say that psychosocial factors are not at work in this study, but their effects were not notable.

Similarly, it has been shown that exposure to low level VOCs and other chemical contaminants have the capacity to induce a psychological reaction (i.e. behavioural changes, difficulties concentrating and depression), in sensitive population groups (American College of Physicians 1989, Marchant 1992, Maroni *et al.* 1995). This study highlighted a similar finding because 34% of the study population reported a variety of psychological symptoms in buildings that had TVOCs ranging from 0.012-1.934 mg/m^3. Psychological symptoms (e.g. difficulties concentrating and light and heavy headedness) were identified and statistically significant (r=.8266) in both the low (<0.20 mg/m^3) and the high (0.20-3.0 mg/m^3) TVOC ranges. Therefore, it appears from the results that VOCs may be a pollutant capable of inducing a psychological reaction in building occupants even at low levels. But a further study would be needed to determine whether it is VOCs, another pollutant, or a combination of contaminants which are causing symptoms of a psychological nature.

The results of the questionnaire survey used in this research highlighted some possible reasons for the distinction in symptom reporting amongst groups of individuals. Overall, many specific factors have been suggested or shown (Brain *et al.* 1988) to be associated with differences in susceptibility, and among these is gender. In this Tasmanian study the gender of subjects were almost equally distributed (104 males and 102 females), but results reveal that the female sex has been disproportionately affected by onset of symptoms. Females (mean

rank=149.05) significantly experienced more SBS symptoms while in the work environment than males (mean rank=115.34). Other studies discuss similar findings (Brain *et al.* 1988, Chandrakumar *et al.* 1994, Ooi *et al.* 1998, Reinikainen *et al.* 1997, Soine 1995, Stenberg & Wall 1995) and claim that females have 2.5 times higher symptom rates than men (Mikatavage *et al.* 1995), and are indeed more susceptible to indoor pollutants.

Age has also been noted as influencing sick building syndrome susceptibility. When reviewing the literature it is believed that SBS symptoms are more prominent in the younger population than any other population group (Smedje *et al.* 1997). Once again the findings of this study are in contrast to this statement because individuals in their middle age (38-47 yrs) consistently reported more symptoms than any other age group (18-27 yrs, 28-37 yrs, 58-48 yrs) in the study sample.

It has been demonstrated, in other studies, that air was perceived as less acceptable with increasing temperature and humidity (Fang *et al.* 1998). Symptoms such as dry throat and blocked nose have been reported significantly where evidence suggested problems with temperature regulation (Chandrakumar *et al.* 1994). In this study, temperature had no observable effect on symptoms. These results complement Valicenti and Wengers (1997) findings which also indicated no observable effects.

Other factors, such as a history of allergenic disease and temporal variations, expectedly affected SBS symptom reporting in this study. Increases in symptoms appeared to be significantly related to the winter months and a history of allergies or asthma.

Winter symptoms may have been caused by pathogens or microbial organisms circulating in the HVAC system. Buildings are heated in the colder seasons, therefore providing a good medium for the proliferation of biological pollutants.

A history of allergies signifies a weakness in immune function, therefore these susceptible individuals (such as those found in this study) have a predisposition to react to allergens found in the indoor environment. The same principle applies to asthma where exposure to airborne allergens can either act as a allergen or as a irritant. According to Maroni *et al.* (1995), immunologic specific IgE sensitisation to an airborne allergen is a major component for this disease, but non-specific hypersensitivity is also important for the asthmatic attacks occuring on exposure to irritants in indoor air. Dust mites, plants, VOCs and moulds are all causative agents which have been proven to induce immunological sensitisation (Maroni *et al.* 1995).

Overall, when account has been taken of such factors (psychosocial, past disease history, age and sex) in other studies, there still remain considerable differences between reported health problems in sick buildings when compared to their healthy counterparts (Skov *et al.* 1989, Wilson *et al.* 1987). This can be

illustrated in the findings of this study where SBS levels significantly differed among the eleven sampled buildings and the control even when psychosocial, age and sex differences were taken into consideration. Occupants in the control building ("healthy") reported no symptoms (that can be attributed to the indoor work environment) during the 10 week sampling period even though subjects were categorised (sex - female, age - 18-27 yrs and past disease history - allergies/hayfever) as being "predisposed" to SBS.

Large scale questionnaire surveys, which have been used extensively in this study, are now common in epidemiological investigations and SBS research, especially in diagnosing and assessing exposure/effect relationships. Their use is primarily because there is no place for any specific medical examination for symptomatic workers. The most pertinent issue centres around the subjective nature of the symptom reporting when using a qualitative method. It is without doubt that a certain level of error would have occurred in this study because of the difficulty in quantifying the accuracy of recalling past symptoms (e.g. symptoms experienced during the last week), even though it has been demonstrated that individuals are very acurate in self diagnosis (Mendell 1993). Therefore it must be taken into consideration that the symptoms recorded by building occupants may not be a precise representation of SBS levels in Tasmanian offices (either by over estimating or under estimating symptom numbers), even though steps were taken to ensure reliability, validity and quality assurance of the qualitative information.

11.2.3.2 INDIVIDUAL EFFECTS

It is believed that SBS is likely to be of multifactorial origin where VOCs play an important part. It has been shown that the presence of VOCs increase inflammatory reactions in the airways as well as numerous other irritative symptoms (Fisk & Rosenfeld 1997, Wieslander *et al.* 1997). The results of this study highlighted similar findings where irritative symptoms, such as those of the eyes, nose, throat and skin, were correlated with certain individual VOC species like 1,1,1-trichloroethane, limonene and ethanol, but no tests were done with these species alone.

Likewise it has been demonstrated by Lundberg (1996) that changes in mood cognition, behaviour, general irritation and nervous system deficits are evident in the presence of low level organic compounds which were not over acceptable standards. The individual VOC concentrations found in the quantitative analysis of this Tasmanian study were all well under current occupational health and environment standards, yet they appeared to induce general irritation in building occupants even at these low concentrations

As anticipated, the results in this study illustrated an association between low level compounds and specific health effects. Although the outcomes of this

need to be further examined using toxicological methodologies, it still provides an interesting result in a systematic building survey. A review of the literature provided some insight into findings identical to those of this study. Norback *et al.* (1990b) for example, observed that symptom prevalence was not only associated with log TVOC values, but also with the concentration of terpenes and the alkane species of hydrocarbons. Similarly, Norback (1995), stated that reported symptoms were related to concentrations of VOCs including xylene, limonene and butanols. This Tasmanian study reflects the findings of both Norback *et al.* (1990b) and Norback (1995), where symptom prevalence was associated with log TVOC values and VOCs species belonging to the alkane family, xylenes, and terpenes.

Alternatively, it may not be the subthreshold levels of VOC species themselves but the reactions between compounds (influenced by oxidative and reactive species) that can generate potential products that are known to be irritants e.g. methacrolein and nonanoic acid (Weschler & Shields 1997, Wolkoff *et al.* 1997). This view may explain why the sick buildings identified in this study have higher SBS sysmptom rates than the control. The sick buildings would have had more reactive species forming potential because of the presence of 15 VOCs (identifiable species in sick building). This is in contrast to the control which had five identifiable species. To extend on this further, as the amount of VOCs increased so did the degree of agonism, which was illustrated in building B10. In summer B10 had 12 detectable VOCs, and retrospective symptom reporting rates were 62%. Winter values showed elevated sypmtom rates of 88.2% and 15 detectable VOC species. This result compares favourably with a study undertaken by Comettomuniz *et al.* (1997), where increasing VOCs suggested increases in symptomatology.

It appears from the results of this study that low level individual VOCs are producing various health effects on a sample population believed (not medically proven) not to be suffering from any clinically diagnosed sensitivity such as MCS. If low level VOCs are having such a noticeable impact on these individuals, it would be interesting to investigate how individuals such as those intolerant to environmental chemicals (Meggs 1997), and those with immune system deficits, react to these low level concentrations.

It is also interesting that all of the VOCs found in this study are either confirmed, probable, or suspected carcinogens, and at high concentrations are categorised as highly toxic or toxic to humans. The toxicity classifications and exposure standards currently applied are for compounds at higher concentrations, but it would be worth examining the health effects to these substances at low concentrations. Perhaps low level VOCs have significant impacts (yet undefined) on health because of the chronic exposures individuals encounter on a daily basis. This could be tested by using a combination of ambient air quality monitoring of VOCs in combination with pathological testing.

11.2.4 DISCUSSION OF AIM FOUR - *To test a relationship between total volatile organic compound levels (TVOC) and work related morbidity*

The range of TVOCs found are representative of studies undertaken elsewhere. This is illustrated in table 11.2, which shows the similarity between TVOC concentrations quantified in this study and other systematic building investigations. Overall, increases in TVOC levels were correlated with both SBS and irritative symptoms in this study but the correlation was weak (r=.031). These results are highlighted by other investigations like Hodgson *et al.* (1991), Hodgson *et al.* (1992), Norback *et al.* (1990a), Norback *et al.* (1990b), and Sundell *et al.* (1993).

TABLE 11.2

Range of TVOC concentrations (mg/m^3) determined in Tasmanian sampled buildings compared to TVOCs from other studies
(Source: Godish, 1995)

Source	TVOC range
This study	0.012-1.93 mg/m^3
Skov *et al.* 1990	0.43-2.63 mg/m^3 (charcoal sampling)
Skov *et al.* 1990	0.1-1.2 mg/m^3 (tenax sampling)
Norback *et al.* 1990b	0.70-0.180 mg/m^3 (primary school buildings)
Norback *et al.* 1990b	0.05-1.38 mg/m^3 (11 building investigation)
De Bortoli *et al.* 1990	0.22-3.93 mg/m^3

Norback *et al.* (1990a), found significant positive correlations with SBS and the logarithmic value of TVOC. Berglund *et al.* (1990) noted the highest association and found that the correlation bewteen the prevalence SBS symptoms and 34 VOCs were highly significant (r=0.96). These findings are representative of some published building studies examining organic compounds (Berglund *et al.* 1990, Norback *et al.* 1990b, Sundell *et al.* 1993). Logarithmically transformed total volatile organic compound and symptoms combined showed a weak positive association (small correlation coefficient) in this study. The scatterplot of these results indicated that the relationship between the two variables was nonlinear because all points did not fall exactly on the regression line. These findings are similar to Lewtas *et al.* (1997), who found that after a certain level, PAHs at low to moderate concentrations were significantly correlated with expsoure but not linearly. In contrast, Norback *et al.* (1990a) and Norback *et al.* (1990b) found that logarithmically transformed TVOCs and symptoms highlighted a linear

relationship. This was shown to be consistent with mouse toxicological studies which demonstrate that sensory irritation is a log linear phenomenon (Alarie 1981).

In contrast, the Danish Town Hall Study (Skov *et al.* 1990, Skov & Valbjorn 1990) and the Californian Healthy Building Study (Fisk *et al.* 1993) and Fanger *et al.* (1988), showed poor correlations between TVOCs and subjective evaluations. In some instances the association was diminished in the presence of strong irritants and formaldehyde. If formaldehyde (not measured in this study) and other strong irritants have the capability of diminishing associations between SBS and TVOCs, then its potential influence on the results of this study have to be considered.

When examining the TVOC concept, Molhave (1995) states that in TVOCs below 0.2 mg/m^3, no effects would be expected in the building population. The findings of this study challenge this tentative relationship because statistical analyses indictated that even at this level, discomfort and health effects were evident. This is especially the case with irritative symptom reporting. According to Molhave (1990), the frequency of general type symptoms, such as headache, in concentrations less than 3 mg/m^3 is due to the interaction of other exposures or the effect of longer exposure durations typical of office environments. This scenario may explain why general manifestations were so frequently reported in buildings even though TVOC levels were consistently less than 3 mg/m^3. The reasons are as follows:

1. The sampled population received long exposures to VOCs which are indicative of the office environment (a typical work day of 8hr, five days a week on average); and
2. The results indicated that TVOCs were associated with another variable (temperature) which has been postulated (by Molhave 1990) to increase the onset of symptoms.

To extend on this, Molhave goes on to say that in the range of 0.20 - 3.0 mg/m^3, both irritation and discomfort would be expected to occur if other factors such as humidity and building variables interact with TVOC exposures. Results indicated that aside from temperature (which only had an minor effect on TVOC values), these other variables had no significant influence on TVOCs and symptomatology.

11.2.5 DISCUSSION OF AIM FIVE - *To examine both the "Total Volatile Organic Compound" concept and low individual concentrations of hydrocarbons as a generic indicator of sensory irritation*

Although the TVOC concept has been used as a generic indicator of sensory irritation (Molhave *et al.* 1997), its use has been cautioned as an indicator of the

risk of nonspecific sensory irritation to non reactive VOCs within a limited range of vapour pressures (Molhave & Nielsen 1992).

The ambiguity of the TVOC concept has been highlighted in the results of this study particularly when examining the influenced of TVOC exposure on sensory irritation. Application of the TVOC ranges was unsuccessful in determining sensory irritation because 40% of irritative symptoms were reported in TVOC levels of below 0.20 mg/m^3. Therefore the term commonly refered to as the "no effects" range should be re-evaluated and perhaps termed as the range in which "limited sensory effects" are expected. Alternatively, it may be necessary to consider the effects of TVOC sensitivity. According to Molhave (1991) and Kjaergaard *et al.* (1991), test subjects who complained of experiencing SBS symptoms prior to controlled TVOC exposures were apparently more sensitive than normal subjects. It may be possible that the subjects in this Tasmanian study were already TVOC "sensitive" (buildings recorded as having a presence of SBS) and therefore recorded sensory effects in the "no effects" range. Or alternatively, irritative symptoms could have been caused by a factor other than TVOC such as moulds, bacteria, SVOCs, VVOCs, electromagnetic fields etc. Another possibility is that daytime TVOC levels were higher than when the building was not occupied. If this was the case then the measurements taken in this study might have underestimated the exposure to building occupants.

Individual exposures to TVOCs that contained certain compounds appeared to have a more significant impact on sensory irritation. This was especially the case for the compounds 1,1,1-trichloroethane, ethanol, 1,2,4-trimethylbenzene, limonene and *n*-butane. These compounds have been documented in causing sensory irritation but an exact cause and effect relationship would require further quantification and perhaps an exposure study rather than a systematic building investigation (such as this Tasmanian study).

Weaknesses in VOC characterisation do exist in all studies, including this Tasmanian study, for example VOCs of microbial origin (Menzies *et al.* 1998) could be causing the sensory irritation (as seen in allergic fungal sinusitis) (Noble *et al.* 1997) reported in the qualitative analysis. Sources are numerous including fungal spores and dust mites. In addition, difficulties arise regarding the complexity of sensory mechanisms, and its associations with other symptoms. Molhave (1990), suggests that headaches can occur as a result of stress in attempting to overide unwanted sensory information, but may not occur as a result of trigeminal nerve stimulation. It is believed that SBS symptoms may arise from multisensory deprivation of signals important to optimal levels of sensory variation (Berglund *et al.* 1984). As a result, variables in the indoor environment lose familiar stimulus patterns, resulting in senory confusion and interpretable sensory signals. Therefore it is hard to know in this Tasmanian study whether feelings of discomfort (headache and fatigue) can be truly classified as "general effects"or whether they

should be included as part of the senory irritation category. It is also unclear whether secondary effects (apnea) from trigeminal nerve stimulation can be termed as true sensory irritation.

11.2.6 DISCUSSION OF AIM SIX - *Review current standards and guidelines in relation to low dose exposures to volatile organic compounds in non-industrial environments (offices)*

The concentrations of the chemical species analysed were unable to be compared with limit values described in relevant guidelines for occupational environments because results represented the mean values for the sampling week. Therefore NOHSC, ACGIH, OSHA and NIOSH standards, as well as the relevant TWAs and STELs were not applied to the study results. If the average TVOC results in this study are any indication, TVOC concentrations have, on many occassions, exceed the Australian guideline value for TVOCs (<500 $\mu g/m^3$ one hour average). What is difficult to determine from the study result is if the TVOCs sampled were constantly at a high concentration or whether a high concentration caused by a sporadic or isolated event (e.g. peaked at one time during the course of the week) while the rest of the sampling was at much lower concentrations.

On the other hand, the advantage in using the quantitative method outlined in this study is that results are more representative of exposures over a working week rather than samples taken for only 15 minutes or 8 hr. In addition, such short term measuring may highlight conditions that are exceptional rather than what conditions are usually like over the long term. These short term testing procedures are more suitable for industrial situations where exposure concentrations have a greater chance of reaching toxic levels.

The measurement of average concentrations over a longer time (i.e. week) in combination with short term testing, generally give a more realistic picture of exposure patterns, especially to the low level VOCs encountered in non-industrial environments like the offices sampled in this survey. These two sampling procedures combined would then be suitable in identifying the probability of disease (risk assessment) with alot more accuracy than if only one method is used.

It appears, when reviewing the literature, that neither tentative TVOC guidelines or standards on individual substances are applicable to non-industrial environment such as offices. The standards and guidelines simply do not address low level chronic exposures to environmental chemicals, and neglect to consider synergistic, low level, and multi contaminant environments.

Because of the potentially serious impacts on health of individuals in the indoor environment, there needs to be a reassessment of current standards and guidelines, and relevant alternatives derived. Even in the absence of complete scientific understanding of indoor air pollution, prudent policy should dictate that

reasonable efforts be made to reduce people's exposure to potentially harmful contaminants. Under current Australian laws, no such policy exists for non-industrial indoor air.

It would seem appropriate to suggest that Australia should re-evaluate its guidelines and activities in relation to air pollution by focusing more on non-industrial indoor air (especially offices). Criteria for acceptable air quality have existed for many years for the industrial work place, some aspects of non-industrial indoor air (e.g. thermal comfort and air exchange rates) and outdoor environments, but for too long the indoor environment has been ignored and illogically viewed as distinct from general air pollution. In reality indoor air pollution has probably more to do with factors such as illhealth and workplace absenteeism, given the very high proportion of time we spend indoors. The approaches taken by Federal governments and organisations in other countries, such as the USEPA, European Union, and WHO, could provide a model for Australia in how to set up our own program for dealing with indoor air pollution.

11.2.7 DISCUSSION OF AIM SEVEN - *To outline and evaluate approaches used in reducing volatile organic compound concentrations in the indoor environment*

As there is such an overwhelming lack of information on organic pollutants in Australian buildings (both commercial and domestic), a focus on the determination of chemical compounds in the air must be considered. To date, characterisation of indoor air pollutants, especially VOCs, has been overlooked and undervalued. As we spend so much time indoors, health effects from potential consumer products and building materials warrants attention. This is even more applicable to compounds identified as confirmed or potential carcinogens.

Despite the fact that the use of organic compounds is ever increasing, some indoor environment strategies can be applied to reduce exposures of people to materials and harmful substances containing VOCs. To complement this some suggestions have been developed during the course of this research and are largely the views of the author. These are:

1. To introduce effective research and development programs to achieve a more complete understanding on indoor air chemistry and toxicology. Generally, there is a paucity of literature on organic compounds especially regarding VVOCs, SVOCs and POMs. The scarcity of data includes a variety of reasons including limitations in analytical procedures, detection difficulties and reactivity of compounds. Therefore the availability of information is greatly reduced and makes interstudy comparisons difficult (a problem frequently cited in this thesis);

2. To determine VOC concentrations typical of office and home environments in Australia, and compare and contrast them to studies found elsewhere. At present very few VOC studies have been undertaken in Australia when compared to other countries and any Australian information on indoor air pollutants, like the results presented in this Tasmanian study, would contribute to Australia's general knowledge on indoor contaminants and their resultant health effects. Because of this overall scarcity of information is difficult to draw conclusions in this study particularly when attempting to compare/contrast results from interstate;
3. It would be beneficial to establish an Australian database of VOCs, so that there is some reliable information on the concentrations and types of VOCs commonly found in Australian buildings, especially those (e.g. VOCs possessing carcinogenic properties) to which the population is likely to be subjected. This must include both systematic building investigation approaches and exposure studies using standardised techniques so direct comparisons can be made to other studies;
4. To evaluate sources of VOCs and systematically find ways of reducing or removing their presence. As outlined in this study the sources of VOCs are extensive, and require ongoing research to determine the full range of sources. Once this has been done, their use in indoor environments should be kept to a minimum. For example, consumer and commercial products, like the cleaning fluids discussed in this study, could be changed to a more chemically inert variety e.g. botanically based (even though this can on occassions cause problems as well) cleaning fluids containing no petrochemicals;
5. To determine ways of promoting public awareness of indoor air pollution, which is an important step in ameliorating conditions. When approaching building managers and owners in this study, the majority were not well informed of the potential hazards of the non-industrial indoor environment, even though they may have had SBS problems in the past. This can be undertaken by establishing community programs and occupational health and safety workshops on indoor air issues. When evaluating awareness of the indoor environment (in office populations) for this thesis it was found that some organisations and groups e.g. CPSU have already taken this step;
6. To introduce compulsory indoor air audits which would assist in ensuring that buildings are maintained, and evaluated to identify any emerging problems. It was found in Tasmania (when researching for background information for this thesis) that there are no mechanisms for indoor assessment as a preventative measure. Only when situations reach a

critical status are they then addressed. This can only be implemented with governmental support at both the State and Federal levels;

7. To introduce preventative forms of action, such as source management strategies, design intervention and remedial actions, using non-regulatory and regulatory tools;
8. To design and implement standards, guidelines and guides that are applicable to non-industrial work environments such as offices and homes. During the course of this study many of the available Australian and overseas standards were not relevant to the low VOC concentrations detected in the results. This makes assessment of the toxicity of the indoor environment difficult, especially when attempting to determine health effects; and
9. To provide information and incentives for action to product manufacturers, architects, engineers, builders, building owners and managers, and building occupants.

If these steps are taken, then the potentially serious impacts on health are greatly reduced.

11.3 Discussion of Hypotheses

The results of this study have highlighted that organic gases are conducive to general illhealth and sensory irritation. Aromatic and aliphatic hydrocarbons appear to be important indoor pollutants. They are not only the most frequently detected, but the ones most likely to provoke sensory irritation and a key pollutant in SBS. Even though other variables were affecting symptoms, such as past disease history, the effects of VOC exposure were evident.

The effects caused by low level volatile organic compounds appear to have been underestimated. Whether individually or in mixtures, evidence from this study suggests that organic compounds, especially those from aromatic and aliphatic families, have the potential to cause minor or debilitating health effects. It also poses the question as to what would the long term effects be from prolonged exposure to these very low concentrations? In reality, exposure does not cease in the work environment, but instead continues in a cycle as the individual passes from one indoor environment (work) to another (home). Measuring the work environment is just one part of the total exposure picture.

Chemicals detected have been proven by other research to be carcinogenic, potentially carcinogenic or toxic given large doses. But it is important to consider that the health effects caused by exposure (in a low level multicontaminant

environment) may not be immediate, instead certain chemical contaminants may result in delayed toxic effects, or accumilative effects.

Some of these questions are beyond the scope of this thesis, but results revealed pose a new set of questions which need to be investigated to determine further the strength of the relationship bewteen VOCs and illhealth.

11.4 Limitations to This Study

Analytical limitations to the study focused on the method used in the GC/MS. Some uncertainty arises because only 1/30,000 of the solvent solution is injected into the GC/MS. Exothermic reactions in the charcoal tubes during the infiltration of samples with carbon disulphide, caused the aromatic hydrocarbon fractions to evaporate. The exact concentration of evaporated aromatics, during the procedure involving the infiltration of carbon disulphide, was not quantified, but it is a commonly used method.

The passive VOC sampling methods used gave average concentrations of VOCs over 7 days. The disadvantage in using this technique is that NOHSC, ACGIH, OSHA and NIOSH standards as well as the relevant TWAs and STELs could not be applied to results. To overcome this, it may be more suitable to combine both long term sampling (weekly averages) and short term sampling (average over one working day) techniques. In addition, because of the passive sampling methods, it was not possible to define maximum and minimum concentrations. Drahonovská and Gajdos (1997) also found this to be the case when comparing the results of passive and active sampling methods. They state that average concentrations of TVOCs obtained using passive techniques are underestimated when compared active sampling methods.

In the majority of cases, data collected was not normally distributed and nonparametric tests had to be used as an alternative technique. Unfortunately these tests have been known to be less powerful than their parametric counterparts.

Some bias in the methodology and statistical analyses would have occured during the sampling because buildings were selected from a list of buildings known to suffer from SBS. In addition all subjects near to the sampling device were included in the survey, which reduces the reliability and validity of results because they were not a random sample. But the use of this technique was neccessary in order to fulfil the objectives of this study.

Specifying the sources of the hydrocarbons was also difficult due to contaminants outgassing from various sources and the infiltration of contaminants from outdoors. TVOC levels could also have been correlated with other variables, for example formaldehyde, particulates and MVOCs, that were not measured in this study.

11.5 Conclusion

Overall, the results of this systematic building investigation highlight interesting results that confirm many of the findings in previous published studies. Tasmania undoubtedly has high SBS prevalence rates, the precise reasons of which are not completely clear. VOCs appear to be a significant contributor, but other variables could also be influencing symptoms. Because of financial and methodological constraints these additional variables could not be measured. The examination of VOCs have been useful in determining background levels commonly found in Tasmanian office environments, and are consistent with many other studies. The use of the TVOC concept has been challenged by the findings of this thesis, but it appears that TVOC categories have not been very useful in determining a buildings potential for SBS. Overall, it appears that VOCs are influencing the health of building occupants significantly. What is required is a further investigation using a variety of additional techniques to confidently assess the extent to which VOCs are impacting occupant health and SBS.

CHAPTER TWELVE

CONTROL of VOLATILE ORGANIC COMPOUNDS in the INDOOR ENVIRONMENT and CONCLUSION

12.1 Introduction

Acknowledgement of the significance of indoor air pollution has progressively developed over the years, and now there is a general acceptance by building owners/managers and building occupants, that problems exist. We can no longer treat the built environment as a neutral factor when analysing work environments, like the offices examined in this study. This chapter therefore discusses some preventative forms of action and concludes the findings of this Tasmanian study. To support the discussion throughout this chapter, references are made to other studies and information found in the indoor air quality literature.

A review of the scientific literature shows sound evidence that VOCs are one of the most likely causes of health effects and comfort problems in the indoor environment. The key is to minimise any future problems by greatly reducing indoor exposure. This way we are dealing with the cause rather than the end result. To accomplish this, emerging and existing technologies can be used in building design, construction and maintenance to create an acceptable and productive work environment.

12.2 Building Design, Construction and Maintenance

All indoor spaces are contaminated with both gas/vapour and particulate phase substances. Sources of these contaminants are numerous, and complete elimination is unachievable, but with some forethought, their presence can be greatly reduced.

Sound building design is the first step in attempting to reduce VOC contamination. Building design includes all elements associated with buildings including site planning, overall architectual design (openable windows), ventilation and climate control, material selection, and the actual construction process (Maroni *et al.* 1995). On reviewing the buildings used in this study, there were many examples of design features (especially the partitions and extensive use of synthetic furniture) which have the potential to outgas volatile organic compounds into the indoor atmosphere. Generally it is difficult to choose between more suitable building materials (e.g. chipboard containing less formaldehyde) and conventional products (e.g. regular chipboard containing formaldehyde resin) because of a variety of factors. Pressure to use commercial products is great given the extent to which products are advertised, the overall lack of awareness by individuals of the chemical properties of products and because of the cost involved in using alternatives. In many cases inappropriate building design elements are unwittingly

introduced even though their potential as a contaminant has been well established. An example of this is chipboard furnishing, which is known to outgas formaldehyde but is frequently used regardless.

Finishes used in construction and applied textiles are believed to be some of the most susceptible for the release of any latent VOCs (Martin *et al.* 1998). Alternatively, Reitzig *et al.* (1998) states that modern ecological building materials contain less volatile and less common substances, but with increased indoor persistence which could partly account for the increasing number of complaints in relation to SBS. As a preventative measure many countries have adopted the use of a labelling system for building materials which have primary emissions of VOCs. These systems unify chemical emission testing, modelling and health evaluation (Wolkoff & Nielsen 1996) to determine a material's potential for impacting comfort and health.

Other techniques can used such as generally reducing the use of materials that have high pollutant sources, reviewing product labels, or heating materials to accelerate the outgassing period. This forced outgassing is more commonly refered to as "building bakeout". Off-gassing of VOCs is greatest when materials are new. In order to minimise occupant exposure, building ventilation rates are increased in the initial months of occupation. Buildings remain unoccupied for as long as possible after installation of materials, followed by the significant raising of indoor temperatures combined with increased ventilation. This process increases off-gassing and speeds the removal of VOCs.

Banning a chemical substance is another alternative. If some substances have a good deal of evidence suggesting that they can seriously harm human health, then it should be banned for use in the indoor environment. Subtances such as pentachlorophenol (PCP) and tobacco smoke are good examples.

Maintenence of buildings is also important. Contamination can be avoided by maintaining the operation of building components, like the HVAC system, and regular cleaning methods. According to Greene *et al.* (1997) and Vincent and Pradalier (1997), correct cleaning and maintenance of HVAC systems allows use without any major sanitary problems. Rational cleaning of internal surfaces essentially removes potential airborne pollutants from the building. Although care must be taken that VOC based or aggressive cleaning agents, such as those found in certain consumer and commercial products are not used. The cleaning products can themselves be re-emitted into the room air or may decompose surface finishes, essentially adding to the pollutant load.

As seen in the results of this thesis, compliance with ASHRAE and SA standards did not guarantee a building free from SBS. Generally, ensuring that building practices are adhered to (including following of standards and guidelines currently in use for thermal comfort and HVAC operations) is a good start in maintaining acceptable air quality. The use of current standards/guidelines for

general indoor air parameters and the advent of new standards/guidelines for VOCs (and other factors not yet addressed like MVOCs) can in combination assist individuals involved in building design and maintenance, by providing procedures and methods for documenting, evaluating, and verifying indoor air parameters. Unfortunately, there are no specific design criteria and standards for VOCs, but the above mentioned design and maintenance protocols should bring about substantial reductions in human exposure to the entire range of indoor air pollutants including those of the volatile organic compound class.

12.3 Economic Implications

The major types of economic cost centre around materials and equipment damages, direct medical costs, workers compensation, and loss of productivity. Legal perspectives in the form of workers compensation claims and cases against building owners/managers are now part of the indoor air arena, especially when exposure to poor indoor air can cause a wide range of health disorders.

Numerous law suits, like those frequently seen in the USA (Ross & Lockey 1994), focus predominantly on adverse health effects or general discomfort resulting from exposure to chemical contaminants. This is particularly pertinent to organic compounds which have the properties of a class "A" carcinogen e.g. tobacco smoke (Ross & Lockey 1994). According to Willing (1996) it is only a matter of time before there is a spate of litigation in Australia against employers, building owners/managers and those involved in the design, construction and fitout of buildings (Weekend Australian 1996). This is especially the case for formaldehyde which is widely used in all aspects of design and fitout of Australian buildings.

According to Fisk and Rosenfeld (1997), discussing the USA situation, direct improvements in the indoor environment and a reduction of pollutants can generate annual savings of US$6 billion to US$19 billion dollars from reduced respiratory disease, and US$1 to US$4 billion dollars in reduced sick building syndrome symptoms. Unfortunately, there are no equivalent Australian estimates, but a proportional economic scenario would not be suprising, given the results of this Tasmanian study and other Australian studies.

12.4 Concluding Statement

By recapitulating the main findings of this thesis, it is clear that volatile organic compounds are having a significant effect on the health of individuals in

non-industrial buildings. In spite of some differences in methodologies, results, both expected and unexpected, can be adequately compared with findings from other countries, which reveal similar results to this Tasmanian study.

Overall, this thesis has provided some much needed information on chemical compounds found indoors and their physico-chemical properties. In addition, it is advancing the science of indoor air quality research in Australia by providing some insight on the dynamics of indoor environments. The results give an overview of VOC concentrations and sick building syndrome levels in selected Tasmanian buildings, as well as providing general information which is applicable for all indoor environments irrespective of their location.

The techniques used in the thesis evaluated conventional IAQ diagnostic procedures in relation to indoor air chemistry, sampling methodology and analyses. The subjectivity of symptom reporting leads to some uncertainty in the accuracy of results, but this is common with all qualitative techniques (particularly systematic building investigations) and not isolated to this study.

The findings contained in this study can clearly be used as a basis for further VOC testing. Results have the potential to be extended on to find general key indicators on which to derive exposure guidelines for both carcinogenic and non-carcinogenic substances. The background levels of VOCs found can be utilised to model the effectiveness of reduction strategies over time. Or alternatively, results can be used as a preliminary data base for other studies to examine and quantify the relationship between VOCs, TVOCs and illhealth. Its use in combination with toxicological studies is one such example.

By evaluating the findings, a major recurring theme emerges in this thesis. That is the overall paucity of published information on organic compounds and their health effects at low level exposures. As with all scientific disciplines, answers to many of the questions will come with time, and with the development of new techniques and methodologies, yielding more accurate results. To extend on this even further, it appears that we have now reached a point where some officially enacted ameliorative action must be implemented to avert illhealth, even though there are gaps in the knowledge of VOCs. The status of office buildings in this Tasmainan study illustrates this, and is a perfect example of unregulated indoor environments, in need of some form of control.

The complex nature of indoor air exacerbates the difficulty in finding one simple answer, but a combination of research, awareness and prospective activities have in combination the potential to improve a very neglected area of air pollution.

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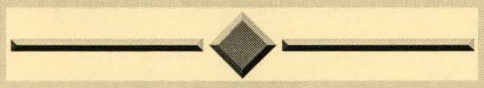

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APPENDICES

APPENDIX 1

INTERNATIONAL STANDARDS, GUIDELINES and CODES RELEVANT to INDOOR AIR QUALITY

1 International Standards, Guidelines and Codes Relevant to Indoor Air Quality

Outlined in this appendix are the International standards, guidelines and codes available for indoor air quality, many of which have been referenced throughout this thesis. The sources of information for this appendix were derived from publications by the following:

1. Maroni, M., Seifert, B. and Lindvall, T., 1995; Indoor Air Quality; Elsevier, Amsterdam, Netherlands;
2. Property Council of Australia (PCA); Building Management Handbook; Sydney, Australia;
3. American Society of Heating and Refrigerating Air Conditioning Engineers (ASHRAE);
4. Australian Building Code (1994);
5. National Occupational Health and Safety Commission (NOHSC:1003)., 1995; *Exposure Standards for Atmospheric Contaminants in the Occupational Environment*; Australian Government Publishing Service, Canberra, ACT; and
6. Web sites - http://www.who.org and http://www.worksafe.gov.au.

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1978 - Principles and Methods for Evaluating the Toxicity of Chemicals, Part 1. Environmental Health Criteria, No 6.

1979 - Sulphur dioxides and Suspended Particulate Matter. Environmental Health Criteria, No 8.

1983 - Guidelines on Studies in Environmental Epidemiology. Environmental Health Criteria, No 27.

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1987 - IARC Environmental Carcinogens. Methods of Analysis and Exposure Measurement. Vol 9, Passive smoking. IARC Sci.Pub, No 8.

1987 - Air Quality Guidelines for Europe. European Series, No 23.

1989 - Formaldehyde. Environmental Health Criteria, No 89.

1990 - Indoor Air Quality: Biological Contaminants. European Series, No 31.

1993 - Indoor Air Quality: a Risk-Based Approach to Health Criteria for Radon Indoors. WHO Working Group, No 108.

WORLD HEALTH ORGANISATION REGIONAL OFFICE FOR EUROPE (WHO/EURO)

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1982 - Indoor Air Pollutants: Exposure and Health Effects. Euro Reports and Studies No 8.

1983 - Indoor Air Pollutants: Exposure and Health Effects. Report on a WHO meeting. Euro Reports and Studies No 78.

1985 - Indoor Air Quality Research. Euro Reports and Studies No 103.

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1987 - Indoor Air Quality: Organic Pollutants. Report on a WHO meeting. Euro Reports and Studies No 111.

1989 - Indoor Air Quality: Organic Pollutants. Euro Reports and Studies No 111.

1990 - Indoor Air Quality: Botanical Contaminants. Euro Reports and Studies No 31.

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AMERICAN SOCIETY OF HEATING AND REFRIGERATING AIR CONDITIONING ENGINEERS (ASHRAE)

1981 - ASHRAE Standard: Ventilation for Acceptable Air Quality. ANSI/ASHRAE.

1981 - ASHRAE Standard: Thermal Environmental Conditions for Human Occupancy (ASHRAE 55 - 1981).

1986 - ASHRAE Proposed American National Standard: Ventilation for Acceptable Indoor Air Quality (ASHRAE 62 - 198112).

1989 - ASHRAE Standard: Ventilation for Acceptable Air Quality (ASHRAE 62 - 1989).

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH)

1986 - Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th Edition.

1990 - Guidelines for the Assessment of Bioaerosols in the Indoor Environment, No 3180.

1991 - Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th Edition.

1994 - Threshold Limit values for chemical Substances and Physical Agents and Biological Exposure Indices, 1994 - 1995.

NORTH ATLANTIC TREATY ORGANISATION AND COMMITTEE ON THE CHALLENGES OF MODERN SOCIETY (NATO/CCMS)

1989 - The Implications of Indoor Air Quality for Modern Society. NATO/CCMS Report, No 183.

1993 - Indoor Air and Its Impact on Man, Methods of Risk Assessment for the Indoor Environment. NATO/CCMS Report on a joint workshop.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH) - WASHINGTON

1976 - Revised Recommended Asbestos Standard. NIOSH Report No. 77- 169.

1977 - Occupational Exposure Sampling Strategy Manual. NIOSH Report No. 77 - 173.

1987 - Guidance for Indoor Air Quality Investigations.

1989 - NIOSH Indoor Air Quality Selected References and Investigations 1971 - 1988.

1990 - NIOSH Manual of Analytical Methods, 4th Edition. NIOSH Report No. 94 - 113.

INTERNATIONAL STANDARDS ORGANISATION (ISO)

1983 - Air Quality - Particle Size Fraction Definitions for Health related sampling, (ISO - TR7708).

1984 - Moderate Thermal Environments - Determination of the PMV and PDD Indices and Specification of the Conditions for Thermal Comfort, (ISO - 7730).

COMMITTEE ON INDOOR AIR QUALITY

1987 - Policies and Procedures for Control of Indoor Air Quality in Existing Buildings.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY (USEPA)

1978 - The Effects of Home Ventilation Systems on Indoor Radon - Radon Daughter Levels. EPA Report No. EPA - 520/ 5-77-011.

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1980 - Air Quality Data 1979 Annual Statistics. EPA Report No. EPA - 450/4-80-014.

1982 - Air Quality Criteria for Oxides of Nitrogen. EPA Report No. EPA - 600/8 - 82 - 026F.

1982 - Air Quality Criteria for Particulate Matter and Sulphur Oxides. EPA Report No. EPA - 600/8 - 82 - 029.

1983 - Health Assessment Document for Acrylonitrile. EPA Report No. EPA - 60/8 - 82/007F.

1986 - Radon Reduction Techniques for Detached Houses, Technical Guidance. EPA Report No. EPA - 625/5 - 86/019.
1986 - Guidelines for Carcinogenic Risk Assessment. No 51, Federal Register 33994.
1989 - Report to Congress on Indoor Air Quality , Vol 1: Federal Programs Addressing of Indoor Air Pollution. EPA Report No. EPA - 400/1 - 89/001B.
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1989 - Report to Congress on Indoor Air Quality , Vol 3. EPA Report No. EPA - 400/1 - 89/001D.
1991 - Introduction to Indoor Air Quality. EPA Report No. EPA - 400/3 - 91/003.
1993 - Review of the National Ambient Air Quality Standards for Sulphur Dioxides: Updated Assessment.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC)

1982 - Chemicals, Industrial Processes and Industries Associated with Cancer. IARC Monograph V1-29.
1982 - Some Industrial Chemicals Associated with Cancer. IARC Monograph V1-29.
1986 - Some Halogenated Hydrocarbons and Pesticide Exposure Associated with Cancer. IARC Monograph V40.

NATIONAL RESEARCH COUNCIL (NRC) - WASHINGTON

1987 - Committee on Indoor Air Quality. Policies and Procedures for Control of Indoor Air Quality in Existing Buildings. Report by NRC.

NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

1975 - Natural Background Radiation in the United States. NCRP Report No. 45, Washington.

DEPARTMENT OF HEALTH AND HUMAN SERVICES (DHHS) - WASHINGTON

1986 - Public Health Service, Office on Smoking and Health. Health Consequences Of Smoking. DHHS (CDC) 87 - 8398.

EUROPEAN CHEMICAL INDUSTRY ECOLOGY AND TOXICOLOGY CENTRE

1986 - A Guide to the Classification of Carcinogens, Mutagens and Teratogens under the Sixth Amendment, Technical Report No 21.

COMMISSION OF EUROPEAN COMMUNITIES (CEC), CONCERTED ACTION GROUP

1986 - Classification and Labelling of Dangerous Substances, Council Directive 79/831/EEC, Sixth Amendment.

1988 - Radon in Indoor Air. Report No 1 Eur 11917 EN.

1989 - Formaldehyde Emission from Wood Based Materials: Guideline for the Determination of Steady State Concentrations in Test Chambers. Report No 2 Eur 12196 EN.

1989 - Indoor Pollution by Nitrous Oxide in European Countries. Report No 3 Eur 12219 EN.

1989 - Sick Building Syndrome - A Practical Guide. Report No 4 Eur 12294 EN.

1989 - Strategy for Sampling Chemical Substances in Indoor Air. Report No 6 Eur 12617 EN.

1990 - Indoor Air Pollution by Formaldehyde in European Countries. Report No 7 Eur 13216 EN.

1991 - Guidelines for the Characterisation of Volatile Organic Compounds Emitted from Indoor Materials and Products Using Small Test Chambers. Report No 8 Eur 13593 EN.

1991 - Effects of Indoor Air Pollution on Human Health. Report No 10 Eur 14086 EN.

1992 - Guidelines for Ventilation Requirements in Buildings. Report No 11 Eur 14449 EN.

1993 - Biological Particles in Indoor Environments. Report No 12 Eur 14988 EN.

1993 - Determination of VOCs Emitted from Indoor Materials and Products. Report No 13 Eur 15054 EN.

NORDIC COMMITTEE FOR BUILDING REGULATIONS (NKB)

1978 - Guidelines for Building Regulations Regarding Acoustic Conditions (NKB No 32).

1984 - Mechanical Ventilation Installation Guidelines (NKB No 52).

1991 - Guidelines for Indoor Climate and Air Quality (NKB No 61E).

DANISH ENGINEERING ASSOCIATION

1981 - Code of Practice for Ventilation Installations (DS 447).

NATIONAL DANSIH BUILDING AGENCY

1986 - Guidelines Regarding Quality Assurance.

1987 - Guidelines Regarding the Division of Responsibility in Construction Work.

1988 - Guidelines Regarding Project Appraisal.

1990 - Guidelines Regarding the Operation of Buildings.

SWEDISH NATIONAL BOARD OF OCCUPATIONAL SAFETY AND HEALTH
1990 - Occupational Exposure Limit Values, Ordinance AFS 1990: 13.

NATIONAL SWEDISH ENVIRONMENTAL PROTECTION AGENCY
1990 - Recommended Values for Air Quality in Urban Areas. Series 90:9.

HOUSE OF COMMONS, ENVIRONMENT COMMITTEE - UNITED KINGDOM
1991 - Indoor Pollution. HMSO (ISBN 0 10 2953910).
1991 - Governments Response to House of Commons Environment Committee. HMSO (ISBN 0 10 1163320).

HEALTH AND SAFETY EXECUTIVE - UNITED KINGDOM
1995 - Occupational Exposure Limits. Guidance Note EH40, HMSO.

DEPARTMENT OF ENVIRONMENT - UNITED KINGDOM
1995 - Expert Panel on Air Quality Standards: Carbon Monoxide.

APPENDIX 2

AUSTRALIAN STANDARDS, GUIDELINES and CODES RELEVANT to INDOOR AIR QUALITY

2 Australian Standards, Guidelines and Codes Relevant to Indoor Air Quality

Outlined in this appendix are the Australian indoor air quality standards, guidelines and codes referenced throughout this thesis. The sources of information for this appendix were derived from publications by the following:

1. National Occupational Health and Safety Commision (NOHSC) 1997; Product and Services Guide;
2. Standards Australia (SA) 1997; Product Guide;
3. Australian Government Publishing Service (AGPS), Canberra;
4. Australia and New Zealand Conservation Council 1994; Guide to Environmental Legislation in Australia and New Zealand;
5. Direct communication with Worksafe Australia and Standards Australia; and
6. Web sites - http://www.worksafe.gov.au and http://www.standards.com.au.

STANDARDS ASSOCIATION OF AUSTRALIA (SAA)

SAAHB23 (1992) - Control Of microbial Growth In Air Handling and Water Systems In Buildings.

STANDARDS AUSTRALIA (SA)

AS1837 (1976) - Code of Practice for Application of Ergonomics to Factory and Office Work.

AS2985 (1987) - Workplace Atmospheres - Methods for Sampling and Gravimetric Determination of Respirable Dust.

AS3580 - Methods for Sampling and Analysis of Ambient Air.

AS2986 (1987) - Working Atmosphere - Organic Vapours - Sampling of Solid Absorption Techniques.

AS666 (1989) - Air Handling and Water Systems of Buildings - Microbial Control.

AS1668.2 (1991) - Mechanical Ventilation for Acceptable Indoor Air Quality.

AS4073 (1993) - Urea Formaldehyde from Thermal Insulation. In Situ Set Foam.

AS3660 (1993) - Protection of Buildings from Subterranean Termites - Prevention, Detection and Treatment of infestation.

AS1668.2 (1994) - Acceptable Ventilation Guidelines.

AS2865 (1995) - Safe Working in a Confined Space.

AS1859 (1996) Part 1, Reconstituted Woodbased Panels: Decorative Overlayed Wood Panels.

AS1859 (1997) Part 2, Reconstituted Woodbased Panels: Particle Board.

AS1859 (1997) Part 3, Reconstituted Woodbased Panels: Medium Density Fibre board

NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL (NHMRC)

1980 - Recommended Radiation Protection Standards for Individuals Exposed to Ionising Radiation.

1982 - Urea Formaldehyde Foam Insulation. Report of 93rd Session.

1983 - Approved Occupational Health Guide: Threshold Limit values (1983 - 1984).

1988 - Australian Guidelines for the Control of Legionella and Legionnaires Disease, AGPS.

1988 - Asthma in Australia- Strategies for Reducing Morbidity and Mortality.

1989 - Code of Practice for the Safe Use of Termaticides.

1989a - Indoor Air Quality. Report of 108th Session, Nov.

1990 - Radon in Houses. Report of The 109th Session, May.

1993 - Volatile Organic Compounds in Indoor Air. Report of 115th Session, June.

NATIONAL OCCUPATIONAL HEALTH AND SAFETY COMMISSION (NOHSC)

1988 - Guide to the Control of Asbestos Hazards in Buildings and Structures [NOHSC: 3002 (1988)].

1988 - Code of Practice for the Safe Removal of Asbestos [NOHSC: 2002 (1988)].

1988 - Guidance Note on the Membrane Filter Method for Estimating Airborne Asbestos Dust [NOHSC: 3003 (1988)].

1989 - Legionnaires Disease and Related Illnesses.

1989 - Guidance Note on the Membrane Filter method for the Estimation of Airborne Synthetic Mineral Fibres [NOHSC: 3006 (1989)].

1989 - Guide on Office Copying Machines [AGPS Cat No 9027571].

1989 - National Strategy for the Prevention of Occupational Skin Disorders [NOHSC: 4002 (1989)].

1990 - National Code of Practice for Safe Use of Vinyl Chloride [NOHSC: 2004 (1990)].

1990 - National Standard for Synthetic Mineral Fibres [NOHSC: 1004 (1990)].

1991 - Exposure Standards for Atmospheric Contaminants in the Occupational Environment.

1994 - Guidance Note on Passive Smoking in the Workplace [NOHSC: 3019].

1994 - Guidance Note for the Control of Workplace Hazardous Substances, Retail Sector [NOHSC: 3018 (1994)].

1994 - List of Designated Hazardous Substances [NOHSC: 10005 (1994)].

1995 - Adopted National Exposure Standards for Atmospheric Contaminants in the Occupational Environment [NOHSC: 1003 (1995)].

1995 - Guidance Note for the Interpretation of Exposure Standards for Atmospheric Contaminants in the Occupational Environment [NOHSC: 3008 (1995)].
1995 - Documentation of the Exposure Standards [NOHSC: 10003 (1995)].
1995 - Workplace Hazardous Substances - Carcinogenic Substances [NOHSC : 1011 (1995)].

THE AUSTRALIAN INSTITUTE OF REFRIGERATION, AIR CONDITIONING AND HEATING INC (AIRAH)

1991 - AIRAH Fire and Smoke Control, Mechanical Ventilation for Acceptable Indoor Air Quality.

AUSTRALIAN BUILDING CODES BOARD

1990 - Building Code Of Australia.
1994 - Building Code Of Australia (Amended version). Amendments 1,2,3,5,and 7.

ACTS WHICH HAVE A RELEVANCE TO INDOOR AIR POLLUTION ISSUES

COMMONWEALTH

National Occupational Health and Safety Commission Act 1985. Function : Development standards, guidelines and promote a national focus on occupational health and safety issues. (Amended by 1989).
Safety Rehabilitation and Compensation Act 1988.
Workers Rehabilitation and Compensation Reform Act 1988. (Reprinted in 1995).
Industrial Relations and Other Legislation Amendment Act 1995. Function : To amend the law about industrial relations, and for other purposes.
Workplace Health and Safety Act 1995. Function : Ensure safe working practices and environments.
Industrial Chemicals (Notification and Assessment) Act (1989). Function : Establishes a national chemicals notification and assessment scheme for use by the public. (Reprinted in 1992).
Environmental Protection (Impact of Proposals) Act 1974. Function : Assessment of the environmental implications of proposals and decisions involving the commonwealth government. (Reprinted in 1990, amended by 1992).

NEW SOUTH WALES

Unhealthy Building Land Act 1990. Function : The EPA may declare land as unhealthy building land. (Amended by 1991).

VICTORIA

Building Act 1993. Function : Regulation of building and building standards.

Environment Protection (Clean Air) Act 1981. Function : Requirements in relation to air pollution.
Health Act 1958. Function : Controls use of lead paint in buildings. (Reprinted in 1992).
Victorian Occupational Health and Safety Act 1985.

SOUTH AUSTRALIA

Development Act 1993. Function : Implements planning, development, design and construction regulations. Includes maintenance of buildings. (Amended by 1994).
Occupational Health Safety and Welfare Act 1986. Function : Controls hazardous substances in the indoor and work environment.

TASMANIA

State Policies and Projects Act 1993. Function : Regulation of environmental standards for air, water and noise.
Public Health Act 1962. : Deals with matters affecting air quality. (Amended by 1982;1983;1984;1985;1989;1990;1991;1993).
Environmental Management and Pollution Control Act 1994.
Workplace Health and Safety Act 1995.

AUSTRALIAN CAPITAL TERRITORY

Buildings (Design and Siting) Act 1964. Function : Controls in relation to siting and construction of buildings. (Reprinted in 1992, amended by 1992 and 1993).
Public Health Act 1928. Function : Protection of public health, especially smoking indoors. (Reprinted in 1991, amended by 1993).
Building Act 1972. Function : Allows the removal of hazardous substances from buildings, like asbestos. (Reprinted in 1993, amended by 1993).

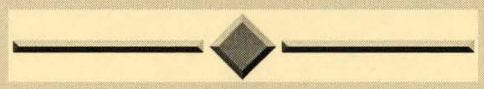
QUEENSLAND

Health Act 1937. Function : Public health with a focus on regulations in relation to smoking indoors. (Reprinted in 1989, amended by 1990;1991;1992;1993).

NEW ZEALAND

Health Act 1956. Function : Provides for public health, includes work conditions, overcrowded buildings etc. (Reprinted in 1987, amended by 1992).
Smoke-Free Environments Act 1990. Function : Reduce the effects of passive smoking in indoor and other environments. (Amended by 1993).

APPENDIX 3



VOCs and the TOXIC EFFECTS ASSOCIATED with EXPOSURE

3 Volatile Organic Compounds and the Toxic Effects Associated with Exposure

Outlined in this appendix are the acute and toxic effects associated with VOC exposure at industrial levels. The appendix also highlights the target organs likely to be affected by exposure. The sources of information for this appendix were derived from publications by the following:

1. Sigma-Aldrich., 1996; Biochemicals, Organic Compounds and Diagnostic Reagents; Sigma-Aldrich PTY LTD, United Kingdom; and
2. Sigma-Aldrich., 1998; Material Safety Data Sheets; Sigma-Aldrich PTY LTD, Sydney, Australia.

Note: Only the volatile organic compounds sampled in this Tasmanian study are listed.

Abbreviations:

RTECS - Registry of toxic effects of chemical substances number.

CONTAMINANT	ACUTE EFFECTS AND TOXICITY	TARGET ORGANS
Benzene C_6H_6 RTECS No: CY 1400000	**Confirmed** human carcinogen. Eye, skin, mucous membrane irritant, nausea, headache, narcotic effects.	Blood,bone marrow,eyes, alters genetic material.
Ethylbenzene C_8H_{10} RTECS No: DA 0700000	**Probable** human carcinogen. Eye, skin, respiratory irritant, causes depression, headche, effects on fertility, fetotoxic, developmental abnormalities.	Sense organs, lungs, behavioural.
Phenol (hydroxybenzene) C_6H_6O RTECS No: RT 5J3325000	**Probable** human carcinogen. Eye, skin, respiratory irritant, mutagenic.	Sense organs, skin, lungs, alters genetic material.
1,2,4-Trimethylbenzene (Pseudocumene) C_9H_{12} RTECS No: DC 3325000	**Probable** human carcinogen. Not enough data currently available.	
Toluene C_7H_8 ($C_6H_5CH_3$) RTECS No: XS 5250000	**Confirmed** human carcinogen. Severe irritation to eyes, skin, Nervous system disturbances, edema, chest pain, ulcerous lesions.	Brain, liver, bladder, kidneys, reproductive hazard.

CONTAMINANT	ACUTE EFFECTS AND TOXICITY	TARGET ORGANS
Xylenes (*m-*, *p-*, *o-*) C_8H_{10} RTECS No: ZE 2275000 (*m-*, *o-*) ZE 2625000 (*p-*)	**Suspected** human carcinogen. Irritating to eyes, skin, lungs, reproductive hazard, narcotic effect, depression, dermatitis.	Nervous system, liver, kidneys.
Styrene (vinylbenzene) C_8H_8 ($C_6H_5CH{:}CH_2$) RTECS No: WL 3675000	**Confirmed** human carcinogen. Heritable genetic damage, mutagen, eye and skin irritant.	Alters genes, central nervous system.
n - Decane (alkane C_{10}) $C_{10}H_{22}$ ($CH_3(CH_2)_8CH_3$) RTECS No: HD 6550000	**Toxic** to humans. Irritating to eyes, skin, lungs, anesthetic action.	Nerves, behavioural.
n - Hexane (alkane C_6) C_6H_{14} ($CH_3(CH_2)_4CH_3$) RTECS No: MN 9275000	Neurotoxic to humans. lung irritant, headache, nausea, cough.	Nerves, behavioural, growth effects.
n - Nonane (alkane C_9) C_9H_{20} ($CH_3(CH_2)_2CH_3$) RTECS No: RA 6115000	**Highly irritative** to humans. Irritation to eyes, skin, and mucous membranes.	Central nervous system.
n - Octane (alkane C_8) C_8H_{18} ($CH_{3(}CH_2)_6CH_3$) RTECS No: RG 8400000	**Toxic** to humans. Irritation in eyes, skin, mucous membranes.	Central nervous system.
Limonene $C_{10}H_{16}$ RTECS No: GW 6360000	**Probable** human carcinogen. Irritating to eyes, skin, lungs, nausea, sensitisation, irreversible effects.	Kidneys, lungs, skin, behavioural.
α - Pinene $C_{10}H_{16}$ RTECS No: DT 7000000	**Toxic** to humans. Severe irritant to mucous membranes, headache, nausea, vomiting.	Tissues, lungs, eyes.
1,2-Dichloroethane $C_2H_4CI_2$ ($ClCH_2CH_2Cl$) RTECS No: KI 0525000	**Probable** human carcinogen. Causes heritable genetic damage, eye, lung and skin irritant, mutagen.	Liver, kidneys, skin, stomach, blood, fertility.
1,1,1-Trichloroethane $C_2H_3CI_3$ (CI_3CCH_3) RTECS No: KJ 2975000	**Suspected** human carcinogen. Destructive to eyes, skin, lungs, narcotic efffect, dermatitis.	Liver,kidneys, heart, behavioural nervous system.
1,1,2,2-Tetrachloroethane (acetylene tetrachloride) $C_2H_2CI_4$ ($CHCI_2CHCI_2$) RTECS No: KI 8575000	**Probable** human carcinogen. Causes heritable genetic damage, impairs fertility.	Eyes, skin, kidneys.

CONTAMINANT	ACUTE EFFECTS AND TOXICITY	TARGET ORGANS
Trichloroethylene C_2HCl_3 ($ClCH{:}CCl_2$) RTECS No: KX 4550000	**Suspected** human carcinogen. Severe irritant to lungs,headache, developmental abnormalities, tumorigenic.	Liver, kidneys, lungs, heart, blood.
Ethanol (alcohol C_2) C_2H_6O (C_2H_5OH) RTECS No: KQ 6300000	**Toxic** to humans. Irritant, nausea, depression, narcotic, tumorgenic.	Liver, nerves, blood, behaviour.
Propanol (alcohol C_3) C_3H_8O ($CH_3CH_2CH_2OH$) RTECS No: T7 7302000	**Highly toxic** to humans. Irritant, depression, nausea, narcotic effects.	Nerves, liver, eyes.
n - Butane (alkane C_4) C_4H_{10} ($CH_3CH_2CH_2CH_3$) RTECS No: EJ 4200000	**Highly toxic** to humans. Causes suffocation, irritant to eyes, lungs, narcotic effects, dermatitis.	Lungs, eyes, brain.
Isobutane (isobutyl alcohol) $C_4H_{10}O$ ($(CH_3)_2CHCH_2OH$) RTECS No: NP 9625000	**Highly toxic** to humans. Causes suffocation, irritant, narcotic effects, dermatitis.	Lungs, eyes, brain.
Carbon tetrachloride (tetrachloromethane) CCl_4 RTECS No: FG 4900000	**Confirmed** human carcinogen. Irritant, stomach pains, nausea, irreversible effects.	Liver, kidneys, nerves, eyes, heart, lungs, behavioural, blood.
Chloroform (trichloromethane) $CHCl_3$ RTECS No: FS 9100000	**Suspected** human carcinogen. Causes heritable genetic damage, Irritant, nausea, dizziness.	Nerves, kidney, fertility, heart.
Acetone C_3H_6O (CH_3COCH_3)	**Toxic** to humans. Irritant, dermatitis.	Sense organs, kidney, brain, lungs.
Ethylene Glycol $C_2H_6O_2$ ($HOCH_2CH_2OH$)	**Toxic** to humans. Irritating to eyes and respiratory system.	Blood, kidneys.

APPENDIX 4

BUILDING INFORMATION QUESTIONNAIRE

4 Building Information Questionnaire

This appendix illustrates the building information questionnaire used in the qualitative analysis for this Tasmanian study.

DATE

INDOOR AIR QUALITY
BUILDING INFORMATION

Building identification and address _____

(1) In which year was the building completed? 1970 s (1) _____ 1990 s (2) _____

FLOORS

(2) what are the floors covered with?

Felt (needle)	☐	1
Carpet	☐	2
Lacquered wood	☐	3
Vinyl (PVC)	☐	4
Linoleum	☐	5
Other	☐	6

Material _____

WALLS

(3) What are the walls covered /treated with?

Wallpaper	☐	1
Paint	☐	2
Wood	☐	3
Untreated textiles	☐	4
Other	☐	5

Material _____

(4) What does the wall consist of (interior side of insulation)?

Bricks	☐	1
Chip board	☐	2
Plaster board	☐	3
Concrete	☐	4
Other	☐	5

Material _____

CEILING

(5) What does the ceiling consist of (inside surface of the room)?

Concrete	☐	1
Plaster board	☐	2
Wood	☐	3
Plaster ceiling	☐	4
Acoustic tiles (perforated plate, mineral wool)	☐	5
Is there an air space above the acoustic tiles? Yes/ No	☐	6
Other ceiling types?	☐	7

Material _____

ROOM DENSITY

(6) Fill in the form for 3 rooms,(include the most, least and typical rooms) numbering the room density per person in m

Room	Area m	Height m	Number of work places	m per person
Most				
Typical				
Least				

(7) Overall rating for room density:

Excellent	☐	1
Good	☐	2
Neutral	☐	3
Bad	☐	4
Very bad	☐	5

(8) Is there any mould growth in the building? Yes/ No ☐

(9) Are there damp spots on the walls/ ceilings/ floors.? Yes/ No ☐

LIGHTING

(10) How are the rooms lit?

By daylight	☐	1
By individual light	☐	2
- fluorescent lamp	☐	3
- incandescent lamp	☐	4
By ceiling light	☐	5
- fluorescent light	☐	6
- incandescent light	☐	7

(11) Is there a solar shading device?

Inside	Yes/No	☐
Outside	Yes/ No	☐

(12) Is there any solar gain during the course of the day?

Yes/ No ☐

HEATING

(13) How are the rooms heated?

Hot water radiators	☐	1
electric radiators	☐	2
Ceiling heat	☐	3
Underfloor heating	☐	4
Warm air heating	☐	5
Other	☐	6

Type _____

(14) How is the heating controlled?

Manual (radiator knob)	☐	1
Thermostat on radiator	☐	2
Wall thermostat	☐	3
Centrally controlled	☐	4
Other	☐	5

Type ☐

(15) Is the air temperature reduced outside working hours? Yes/ No _____

FURNITURE

(16) What is the furniture made of in a typical room? ☐

		% of room consisting of this material
Veneered chip board ☐	1	%
Vinyl ☐	2	%
Metal ☐	3	%
Solid wood ☐	4	%
Other ☐	5	%
☐		

(17) The age of the furniture:

Mainly new	1	_____
Older than three years	2	_____

(18) Are there any curtains/ textiles/ plastic or metal window shading devices?

Yes/ No ☐

(19) When were they last washed / dry cleaned? _____

(20) Are there any large green plants?

Flowering or green leaved plants ☐	1	
- Treated with chlorophyll ☐	2	
- Treated with insecticide ☐	3	
Other ☐	4	

(21) Which rooms have " extensive" use of

		Which rooms? All/Most/Some
Solvents ☐	1	_____
Other chemicals ☐	2	_____
Copying machines ☐	3	_____
Video display units ☐	4	_____
Laserprinters ☐	5	_____
Carbonless paper ☐	6	_____
Animals (stuffed or living) ☐	7	_____

(22) How are the rooms ventilated?

Windows that can be opened ☐	1
By exhaust ventilation ☐	2
By supply air ventilation ☐	3
Other ☐	4

(23) Is smoking generally allowed? Yes/ No ☐

(24) Only allowed in separately ventilated rooms? Yes/ No ☐

(25) How often are the rooms cleaned?

		Daily	2-4 times a week	Once a week	1-3 times a month
Cleaning the tables	1				
Cleaning the walls	2				
Washing the floors	3				
Vacuum cleaning	4				
Mopping	5				
Sweeping	6				
How often is the major cleaning done?	7				
Who is responsible for the cleaning?					

Within the last few years were there any alterations in the cleaning? Yes ☐ No ☐

Which year?

Which kind of detergents are used?

Name and content

AIR CONDITIONING

(26) What type of air conditioning system is in use?

Single package heat pump unit (heating/ cooling)	☐	1
Heat pump split system (heating/ cooling)	☐	2
Reversible air to water heat pump (heating/ cooling)	☐	3
Hermetic centrifugal liquid chiller (cooling)	☐	4
Packaged air-cooled reciprocating chiller (cooling)	☐	5
Other	☐	6

(27) Does the air system use a

Cooling tower	☐	1
Dry cooler	☐	2

(28) Air exchange rate: (outdoor air)

Estimated ☐
Measured ☐

< 0.5 times per hour	☐	1
0.5 - 1 per hour	☐	2
1 - 3 times per hour	☐	3
> 3 times per hour	☐	4

(29) Lowest outdoor air supply per person for a typical room

l/s. person in building/ room

(30) Are the air outlets placed

Close to air inlets	☐	1
High	☐	2
Low	☐	3

(31) Are the exhaust terminals placed

Close to air inlet terminals	☐	1
High	☐	2
Low	☐	3

(32) Is there -

Recirculation?	☐	1 % _____
Humidification?	☐	2 Type_____
Cooling?	☐	3 Type_____
Heat recovery?	☐	4 Type_____

(33) How is the ventilation system operated?

Manually	☐	1
By individually opening windows	☐	2
By automatic control	☐	3

(34) Operating at.....

.....	Full performance	from.....to.....(hour/day)
.....	Reduced performance	from.....to.....(hour/day)
.....	Stopped system	from.....to.....(hour/day)
.....	100% recirculation	from.....to.....(hour/day)
.....	% return air	from.....to.....(hour/day)

(35) Is there an operation and maintenance programme for the ventilation system?

Yes/ No ☐

(36) Who is responsible for operation and maintenance? _____

(37) Have the following components been serviced?

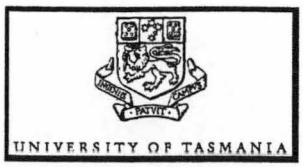
Filter	☐	1
Heating/ cooling batteries	☐	2
Heat exchangers	☐	3
Humidifier	☐	4
Ducts	☐	5
Outlet and exhaust	☐	6
Air terminals	☐	7

APPENDIX 5

SYMPTOM QUESTIONNAIRE

5 Symptom Questionnaire

This appendix illustrates the symptom questionnaire used in the qualitative analysis for this Tasmanian study.



DATE

SAMPLE NUMBER

This is a University research program. The responses will be treated in complete confidentiality.
Any symptom information will not be disclosed nor will the people concerned be identifiable.

BACKGROUND INFORMATION

(1) Year of birth .....

(2) Sex M ☐ F ☐ (Please tick)

Please circle response

		1	2
(3)	When did you start employment at your present workplace ..... (YEAR)		
(4)	Does anybody in your family suffer from any allergies ?	YES	NO
(5)	Have you ever suffered from hayfever ?	YES	NO
(6)	Have you ever suffered from eczema ?	YES	NO
(7)	Have you ever suffered from asthmatic problems ?	YES	NO
(8)	Prior to entering the workplace today did you suffer from any symptoms of illhealth in general.	YES	NO

(9) If yes, what kind of symptoms?

.....
.....
.....

PRESENT SYMPTOMS

	(10) During the sampling period did you experience any of the following symptoms?	1	2
(a)	Fatigue	YES	NO
(b)	Light headedness	YES	NO
(c)	Heavy headedness	YES	NO
(d)	Difficulties concentrating	YES	NO
(e)	Stuffy or runny nose	YES	NO
(f)	Dry or flushed facial skin	YES	NO
(g)	Dizziness	YES	NO
(h)	Headache	YES	NO
(i)	Eye irritation or burning of the eyes	YES	NO
(j)	Metallic taste	YES	NO
(k)	Sinusitis	YES	NO
(l)	Tinnitus (ringing in the ears)	YES	NO
(m)	Joint pains	YES	NO
(n)	Irregular heart rhthym (missed beats, extra beats)	YES	NO
(o)	Nausea	YES	NO
(p)	Apnea (swelling of the airways and nose)	YES	NO
(q)	Cough	YES	NO
(r)	Giddiness (dizziness and nausea and sweaty palms)	YES	NO
(s)	Heartburn	YES	NO
(t)	Euphoric effects	YES	NO
(u)	Irritation of the skin on the hands or face	YES	NO

(v) Flu like symptoms YES NO
(w) Swelling of the liver region YES NO
(x) Swelling of the kidney region YES NO
(y) Myalgia (muscle pains) YES NO
(z) Other ... (please state) ..... YES NO

NATURE OF SYMPTOMS

	1	2
(11) Do the symptoms abate or cease -		
(a) Immediately after you leave your work place	YES	NO
(b) Gradually after you leave your work place	YES	NO
(12) Does it seem to vary with -		
(a) Time of the day	YES	NO
(b) Day of the week	YES	NO
(c) Season of the year	YES	NO

.....

(13) Do the symptoms recur when you return to your work place ?	YES	NO
(14) If yes, do you believe it is due to your work environment ?	YES	NO
(15) Do you regard your work as interesting and stimulating?	YES	NO
(16) Do you have much work to do ?	YES	NO
(17) Do you have any opportunity to influence your work conditions?	YES	NO
(18) Do your fellow-workers help you with problems you may have in your work?	YES	NO

Thank you for your participation.
If you have any more queries concerning this questionnaire, please do not hesitate to contact me.

DESIREE MESAROS ph: (014) 906694

APPENDIX 6

VOCs and EXPOSURE STANDARDS

6 Volatile Organic Compounds and Exposure Standards

Outlined in this appendix are the NOHSC, ACGIH, OSHA and NIOSH standards (TWAs and STELs) for the VOCs examined in this thesis. The sources of information for this appendix were derived from publications by the following:

1. Sigma-Aldrich., 1996; Biochemicals, Organic Compounds and Diagnostic Reagents; Sigma-Aldrich PTY LTD, United Kingdom;
2. Sigma-Aldrich., 1998; Material Safety data Sheets; Sigma-Aldrich PTY LTD, Sydney, Australia; and
3. National Occupational Health and Safety Commission (NOHSC:1003)., 1995; *Exposure Standards for Atmospheric Contaminants in the Occupational Environment*; Australian Government Publishing Service, Canberra, ACT.

Note: Only the volatile organic compounds sampled in this Tasmanian study are listed. No entry indicates that STELs and TWAs were not available for that compound.

Abbreviations:

RTECS - Registry of Toxic Effects of Chemical Substances Number.

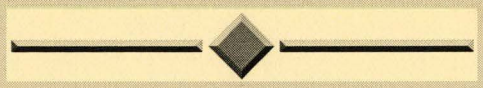
CONTAMINANT	**NOHSC standard**	**ACGIH standard**	**OSHA standard**	**NIOSH standard**
Benzene C_6H_6 RTECS No: CY 1400000 Boiling point 80°C Vapour pressure 20°C - 101hPa	TWA 5 ppm	TLV-TWA 10 ppm	TWA 1 ppm STEL 5 ppm	TWA 0.1 ppm
Ethylbenzene C_8H_{10} RTECS No: DA 0700000 Boiling point 136°C Vapour pressure 20°C - 9.3	TWA 100 ppm	TLV- TWA 100 ppm STEL 125 ppm	TWA 100 ppm	TWA 100ppm STEL 125 ppm

CONTAMINANT	NOHSC standard	ACGIH standard	OSHA standard	NIOSH standard
1,2,4- Trimethylbenzene (Pseudocumene) C_9H_{12} RTECS No: DC 3325000 Boiling point 169°C	TWA 25 ppm			
Toluene C_7H_8 ($C_6H_5CH_3$) RTECS No: XS 5250000 Boiling point 111°C Vapour pressure 20°C - 29 hPa	TWA 100 ppm STEL 150 ppm	TLV-TWA 100 ppm	TWA 200 ppm	TWA 100 ppm STEL 150 ppm
Xylenes (*m-*, *p-*, *o-*) C_8H_{10} RTECS No: ZE 2275000 (*m-*, *o-*) ZE 2625000 (*p-*) Boilng Point 139°C (*m*) 138°C (*p*) 144°C (*o*) Vapour pressure 20°C - 8 hPa (*m*) 20°C - 8.2 hPa (*p*) 20°C - 6.7 hPa (*o*)	TWA 80 ppm STEL 150 ppm	TLV-TWA 100 ppm STEL 150 ppm	TWA 100 ppm STEL 150 ppm	TWA 100 ppm STEL 200 ppm
n - Decane (alkane C_{10}) $C_{10}H_{22}$ ($CH_3(CH_2)_8CH_3$) RTECS No: HD 6550000 Boiling point 174°C Vapour pressure 20°C - 1.9 hPa				

CONTAMINANT	NOHSC standard	ACGIH standard	OSHA standard	NIOSH standard
n - Nonane (alkane C_9) C_9H_{20} ($CH_3(CH_2)_2CH_3$) RTECS No: RA 6115000 Boiling point 150°C Vapour pressure 20°C - 5 hPa		TLV-TWA 200 ppm		TWA 200 ppm
n - Octane (alkane C_8) C_8H_{18} ($CH_3(CH_2)_6CH_3$) RTECS No: RG 8400000 Boiling point 126°C Vapour pressure 20°C - 15 hPa		TLV-TWA 400 ppm	TWA 500 ppm	TWA 75 ppm STEL 385 ppm
Limonene $C_{10}H_{16}$ RTECS No: GW 6360000 Boiling point 177 - 178°C Vapour pressure 20°C - 2.1 hPa				
α - Pinene $C_{10}H_{16}$ RTECS No: DT 7000000 Boiling point 155 - 156°C Vapour pressure 25°C - 5 hPa				

CONTAMINANT	NOHSC standard	ACGIH standard	OSHA standard	NIOSH standard
1,1,1- Trichloroethane $C_2H_3Cl_3$ (Cl_3CCH_3) RTECS No: KJ 2975000 Boiling point 74°C Vapour pressure 20°C - 133 hPa	TWA 125 ppm	TLV-TWA 350 ppm STEL 450 ppm	TWA 350 ppm	TWA 350 ppm
Ethanol (alcohol C_2) C_2H_6O (C_2H_5OH) RTECS No: KQ 6300000 Boiling point 78°C Vapour pressure 20°C - 59 hPa	TWA 1000 ppm	TLV-TWA 1000 ppm	TWA 1000 ppm	TWA 100 ppm
n - Butane (alkane C_4) C_4H_{10} ($CH_3CH_2CH_2CH_3$) RTECS No: EJ 4200000 Boiling point 1°C Vapour pressure 21°C - 1150 hPa	TWA 800 ppm	TLV-TWA 800 ppm		TWA 800 ppm
Isobutane (isobutyl alcohol) $C_4H_{10}O$ ($(CH_3)_2CHCH_2OH$) RTECS No: NP 9625000 Boiling point 12°C Vapour pressure 19°C - 21600 hPa	TWA 50 ppm	TLV-TWA 800 ppm		
Acetone C_3H_6O (CH_3COCH_3) Boiling point 56°C Vapour pressure 20°C - 233 hPa	TWA 500 ppm STEL 1000 ppm	TLV-TWA 750 PPM STEL 1000 ppm	TWA 1000 ppm	TWA 250 ppm

APPENDIX 6.1



FREQUENCY of DETECTED VOCs

6.1 Volatile Organic Compounds Sampled - Fequencies

Outlined in this appendix are some of the results of the statistical analyses referred to in this thesis. The figures represent the frequencies of the volatile organic compunds detected in the study and where possible their (upper (UDL) and lower (LDL)) detection limits (mg/m^3). The sources of information The source of information for this appendix was derived from the following:

1. SPSS ® Program (SPSS Inc. Software 1990).

ISOBUTANE

Mean	.031	Std err	.005	Median	.018
Mode	.012	Std dev	.050	Variance	.002
Kurtosis	37.467	S E kurt	.457	Skewness	5.707
S E skew	.230	Range	.400	Minimun	.000
Maximum	.400	Sum	3.379	LDL/UDL	

***n*-BUTANE**

Mean	.045	Std err	.005	Median	.032
Mode	.023	Std dev	.057	Variance	.003
Kurtosis	51.650	S E kurt	.457	Skewness	6.273
S E skew	.230	Range	.539	Minimun	.000
Maximum	.539	Sum	5.000	LDL/UDL	

ACETONE

Mean	.018	Std err	.001	Median	.015
Mode	.016	Std dev	.013	Variance	.000
Kurtosis	1.231	S E kurt	.457	Skewness	1.240
S E skew	.230	Range	.059	Minimun	.000
Maximum	.059	Sum	2.031	LDL/UDL	25-3600

BENZENE

Mean	.010	Std err	.001	Median	.008
Mode	.005	Std dev	.015	Variance	.000
Kurtosis	80.024	S E kurt	.457	Skewness	8.341
S E skew	.230	Range	.155	Minimun	.000
Maximum	.155	Sum	1.102	LDL/UDL	1.5-9.6

TOLUENE

Mean	.029	Std err	.002	Median	.025
Mode	.025	Std dev	.022	Variance	.000
Kurtosis	19.365	S E kurt	.457	Skewness	3.553
S E skew	.230	Range	.178	Minimun	.000
Maximum	.178	Sum	3.145	LDL/UDL	2-570

ETHYLBENZENE

Mean	.006	Std err	.001	Median	.004
Mode	.003	Std dev	.007	Variance	.000
Kurtosis	6.248	S E kurt	.457	Skewness	2.600
S E skew	.230	Range	.036	Minimun	.000
Maximum	.036	Sum	.649	LDL/UDL	

XYLENES

Mean	.022	Std err	.002	Median	.018
Mode	.016	Std dev	.021	Variance	.000
Kurtosis	18.997	S E kurt	.457	Skewness	3.967
S E skew	.230	Range	.150	Minimun	.000
Maximum	.150	Sum	2.389	LDL/UDL	3-1320

1,2,4-TRIMETHYLBENZENE

Mean	.017	Std err	.005	Median	.006
Mode	.004	Std dev	.048	Variance	.002
Kurtosis	22.192	S E kurt	.457	Skewness	4.757
S E skew	.230	Range	.294	Minimun	.000
Maximum	.294	Sum	1.845	LDL/UDL	

1,1,1-TRICHLOROETHANE

Mean	.036	Std err	.004	Median	.024
Mode	.002	Std dev	.037	Variance	.001
Kurtosis	5.198	S E kurt	.457	Skewness	2.017
S E skew	.230	Range	.210	Minimun	.000
Maximum	.210	Sum	3.995	LDL/UDL	20-3240

α-PINENE

Mean	.004	Std err	.001	Median	.002
Mode	.000	Std dev	.006	Variance	.000
Kurtosis	7.324	S E kurt	.459	Skewness	2.612
S E skew	.231	Range	.033	Minimun	.000
Maximum	.033	Sum	.470	LDL/UDL	

LIMONENE

Mean	.005	Std err	.001	Median	.003
Mode	.000	Std dev	.006	Variance	.000
Kurtosis	22.751	S E kurt	.457	Skewness	4.023
S E skew	.230	Range	.050	Minimun	.000
Maximum	.050	Sum	.500	LDL/UDL	

ETHANOL

Mean	.170	Std err	.034	Median	.081
Mode	.000	Std dev	.274	Variance	.075
Kurtosis	19.544	S E kurt	.586	Skewness	4.054
S E skew	.297	Range	1.758	Minimun	.000
Maximum	1.758	Sum	11.029	LDL/UDL	40-5700

***n*-OCTANE**

Mean	.005	Std err	.002	Median	.004
Mode	.000	Std dev	.005	Variance	.000
Kurtosis	-2.221	S E kurt	1.334	Skewness	.191
S E skew	.687	Range	.012	Minimun	.000
Maximum	.012	Sum	.049	LDL/UDL	8-7050

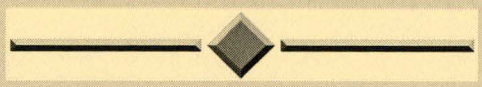
***n*-NONANE**

Mean	.037	Std err	.013	Median	.021
Mode	.000	Std dev	.041	Variance	.002
Kurtosis	-1.781	S E kurt	1.334	Skewness	.415
S E skew	.687	Range	.101	Minimun	.000
Maximum	.101	Sum	.367	LDL/UDL	

***n*-DECANE**

Mean	.052	Std err	.018	Median	.034
Mode	.000	Std dev	.057	Variance	.003
Kurtosis	-1.893	S E kurt	1.334	Skewness	.359
S E skew	.687	Range	.137	Minimun	.000
Maximum	.137	Sum	.521	LDL/UDL	

APPENDIX 7



VOC CONCENTRATIONS DURING the TEN SAMPLING WEEKS

7 VOC Concentrations During the Ten Sampling Weeks

Outlined in this appendix are the results of the quantitative analysis in this Tasmanian study. VOCs listed are the mean weekly concentrations for each compound quantified in all buildings (11 sample buildings and 1 control) during the ten sampling weeks. Concentrations are shown in mg/m^3.

Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B1	0.0493	0.089	0.0156	0.0061	0.0206	0.0026	0.0154	0.0065	0.0504
Week2.B1	0.1141	0.1759	0.0267	0.0252	0.0366	0.0039	0.0224	0.0089	0.0214
Week3.B1	0.0878	0.1482	0.0139	0.0042	0.0153	0.0022	0.0127	0.0059	0.0093
Week4.B1	0.0568	0.0922	0.0207	0.0091	0.0305	0.00396	0.042	0.0089	0.0126
Week5.B1	0.0866	0.1365	0.0443	0.032	0.0912	0.0071	0.0484	0.0124	0.0164
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B2	0.0158	0.032	0.0156	0.0104	0.032	0.0035	0.0149	0.0049	0.0175
Week2.B2	0.0392	0.0568	0.0137	0.0197	0.0443	0.0044	0.018	0.0069	0.0214
Week3.B2	0.0224	0.0426	0.0147	0.011	0.0225	0.00264	0.0149	0.0059	0.0142
Week4.B2	0.0139	0.0279	0.0113	0.0071	0.0187	0.0022	0.0145	0.0059	0.0082
Week5.B2	0.0077	0.0217	0.0046	0.0052	0.016	0.00176	0.0105	0.0025	0.01312
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B3	0.0205	0.0421	0.0241	0.011	0.0279	0.0035	0.0167	0.0079	0.0126
Week2.B3	0.0089	0.0236	0.0193	0.02	0.0359	0.0057	0.0285	0.0095	0.0246
Week3.B3	0.0202	0.0359	0.0113	0.0094	0.0228	0.0031	0.0163	0.0055	0.0142
Week4.B3	0.0147	0.0303	0.0171	0.0097	0.0259	0.0035	0.0189	0.0039	0.0126
Week5.B3	0.0284	0.0431	0.0139	0.0129	0.0229	0.00308	0.0176	0.0059	0.0131
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B4	0.0168	0.036	0.0226	0.0119	0.0378	0.0039	0.0242	0.0075	0.0547
Week2.B4	0.0211	0.0577	0.0158	0.0139	0.0321	0.00484	0.0281	0.0089	0.0805
Week3.B4	0.025	0.0412	0.0166	0.0129	0.0328	0.0044	0.0233	0.0089	0.069
Week4.B4	0.0125	0.0265	0.0099	0.0126	0.0244	0.0022	0.0211	0.0055	0.0416
Week5.B4	0.0069	0.0118	0.0091	0.0029	0.0145	0.0017	0.0129	0.0029	0.0334
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B5	0.0147	0.031	0.0229	0.0148	0.0296	0.003	0.0193	0.0069	0.058
Week2.B5	0.0164	0.0286	0.0125	0.0175	0.037	0.00352	0.0273	0.0075	0.0438
Week3.B5	0.0233	0.0489	0.0291	0.0207	0.0393	0.0049	0.031	0.0124	0.0646
Week4.B5	0.0166	0.0438	0.0219	0.011	0.02214	0.00264	0.0197	0.0049	0.0416
Week5.B5	0.0053	0.0111	0.0048	0.0023	0.0061	0.00132	0.00747	0.0025	0.0241
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B6	0.0219	0.0409	0.0205	0.0159	0.0214	0.0026	0.0167	0.0075	0.0942
Week2.B6	0.0243	0.0445	0.0185	0.01197	0.0252	0.0053	0.0211	0.0075	0.0854
Week3.B6	0.0221	0.0402	0.0125	0.00032	0.0137	0.00264	0.0136	0.0054	0.0882
Week4.B6	0.0149	0.0277	0.0077	0.0022	0.0145	0.0026	0.0114	0.0055	0 0701
Week5.B6	0.0258	0.0409	0.0048	0.0003	0.0252	0.0022	0.01363	0.0035	0.0783
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B7	0.0231	0.044	0.0332	0.0126	0.0393	0.0044	0.023	0.0095	0.0657
Week2.B7	0.0154	0.0306	0.0231	0.0091	0.0359	0.0044	0.0246	0.0085	0.0493
Week3.B7	0.0144	0.0241	0.0089	0.0042	0.0282	0.0031	0.0194	0.0075	0.0515
Week4.B7	0.0152	0.0267	0.0129	0.0084	0.0256	0.0053	0.018	0.0085	0.046
Week5.B7	0.0089	0.0255	0.0274	0 0055	0.0195	0.0022	0.01187	0.0035	0.0476
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B8	0.035	0.085	0.044	0.017	0.034	0.004	0.022	0.006	0.093
Week2.B8	0.043	0.101	0.048	0.155	0.035	0.004	0.026	0.007	0 126
Week3.B8	0.064	0.139	0	0.014	0.036	0.004	0.025	0.005	0.013
Week4.B8	0.32	0.539	0.053	0.019	0.042	0.005	0.029	0.008	0.033
Week5.B8	0.063	0.11	0.046	0.015	0.046	0.005	0.028	0.007	0.032
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B9	0.053	0.05	0.01	0.002	0.009	0.001	0.01	0.007	0.02
Week2.B9	0	0	0.001	0	0	0	0	0	0
Week3.B9	0.08	0.101	0.001	0.003	0.006	0	0	0	0.01
Week4.B9	0.003	0.006	0.019	0.001	0.005	0.001	0.005	0.001	0.083
Week5.B9	0.05	0.063	0.014	0.002	0.05	0.001	0.007	0.002	0.008

Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B1	0.0017	0.0045				
Week2.B1	0.0028	0				
Week3.B1	0.0039	0.0051				
Week4.B1	0.009	0.0056				
Week5.B1	0.0028	0.0113				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B2	0.0022	0				
Week2.B2	0	0				
Week3.B2	0.0.0022	0				
Week4.B2	0.0079	0.0028				
Week5.B2	0.0005	0.0011				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B3	0	0				
Week2.B3	0.0079	0.0034				
Week3.B3	0	0				
Week4.B3	0.0022	0.0023				
Week5.B3	0.0011	0.0017				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B4	0.0022	0				
Week2.B4	0.0085	0				
Week3.B4	0.0062	0				
Week4.B4	0	0				
Week5.B4	0	0				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B5	0.0028	0.0028				
Week2.B5	0	0				
Week3.B5	0.0056	0.0028				
Week4.B5	0	0				
Week5.B5	0	0				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B6	0.0067	0.0022				
Week2.B6	0.01072	0.0056				
Week3.B6	0.0028	0.0051				
Week4.B6	0.0045	0.0034				
Week5.B6	0.0005	0.0056				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B7	0.0152	0.0073				
Week2.B7	0.0079	0.0039				
Week3.B7	0.0039	0.0051				
Week4.B7	0.0164	0.0034				
Week5.B7	0.0011	0.0017				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B8	0.002	0.05				
Week2.B8	0.001	0.002				
Week3.B8	0.004	0.004				
Week4.B8	0.004	0.01				
Week5.B8	0.003	0.019				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B9	0.002	0.005	1.758	0	0	0.007
Week2.B9	0	0	0	0	0	0
Week3.B9	0	0	0.151	0	0	0
Week4.B9	0.001	0.02	0.045	0	0	0
Week5.B9	0	0	0.043	0	0	0

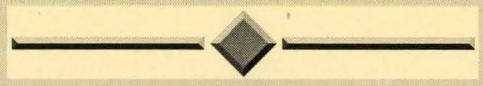
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B10	0.06	0.068	0.006	0.0015	0.013	0.003	0.027	0.012	0.024
Week2.B10	0.029	0.024	0.004	0.008	0.008	0.001	0.0114	0.007	0.003
Week3.B10	0.046	0.052	0.004	0.002	0.014	0.004	0.031	0.0139	0.003
Week4.B10	0.048	0.051	0.007	0.002	0.01	0.002	0.02	0.014	0.038
Week5.B10	0.062	0.068	0.007	0.012	0.013	0.003	0.027	0.018	0.024
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.B11	0.029	0.049	0.021	0.005	0.037	0.021	0.102	0.005	0.002
Week2.B11	0.053	0.09	0.035	0.01	0.042	0.008	0.05	0.004	0.002
Week3.B11	0.03	0.04	0.01	0.001	0.013	0.004	0.01	0.004	0.01
Week4.B11	0.01	0.02	0.007	0.008	0.025	0.005	0.01	0.007	0.03
Week5.B11	0.025	0.03	0.02	0.005	0.05	0.032	0.02	0.004	0.03
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B1	0.025	0.044	0.033	0.008	0.027	0.004	0.021	0.006	0.024
Week7.B1	0.028	0.059	0.057	0.005	0.025	0.003	0.018	0.004	0.018
Week8.B1	0.029	0.04	0.008	0.01	0.025	0.004	0.025	0.008	0.018
Week9.B1	0.02	0.03	0.013	0.006	0.025	0.004	0.024	0.005	0.022
Week10.B1	0.028	0.053	0.023	0.011	0.029	0.003	0.024	0.004	0.03
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B2	0.01	0.021	0.016	0.016	0.044	0.015	0.121	0.032	0.005
Week7.B2	0.005	0.012	0.009	0.009	0.022	0.002	0.005	0.003	0.005
Week8.B2	0.013	0.015	0.008	0.007	0.021	0.003	0.014	0.003	0.002
Week9.B2	0.043	0.067	0.029	0.017	0.058	0.005	0.03	0.007	0.006
Week10.B2	0	0	0	0	0.006	0	0.006	0	0
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B3	0.009	0.026	0.012	0.01	0.023	0.003	0.016	0.004	0.008
Week7.B3	0.01	0.022	0.011	0.01	0.018	0.002	0.013	0.003	0.007
Week8.B3	0.008	0.0175	0.008	0.007	0.021	0.003	0.016	0.004	0.005
Week9.B3	0.017	0.025	0.012	0.007	0.019	0.004	0.017	0.004	0.005
Week10.B3	0.029	0.04	0.018	0.013	0.032	0.005	0.021	0.005	0.009
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B4	0.014	0.038	0.016	0.012	0.026	0.003	0.016	0.005	0.021
Week7.B4	0.025	0.055	0.059	0.016	0.111	0.018	0.15	0.021	0.03
Week8.B4	0.012	0.014	0.004	0.004	0.021	0.003	0.019	0.004	0.09
Week9.B4	0.0146	0.023	0.014	0.007	0.024	0.004	0.019	0.005	0.012
Week10.B4	0.018	0.036	0.016	0.012	0.028	0.004	0.017	0.004	0.014
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B5	0.021	0.018	0.013	0.009	0.025	0.003	0.019	0.004	0.033
Week7.B5	0.034	0.023	0.019	0.008	0.022	0.003	0.017	0.004	0.043
Week8.B5	0.007	0.014	0.006	0.003	0.022	0.003	0.017	0.004	0.024
Week9.B5	0.007	0.014	0.013	0.005	0.025	0.004	0.021	0.005	0.027
Week10.B5	0	0	0.016	0.015	0.029	0.004	0.022	0.004	0.04
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6,B6	0.019	0.024	0.008	0.007	0.034	0.006	0.004	0.006	0.055
Week7,B6	0.015	0.024	0.014	0.023	0.045	0.002	0.007	0.018	0.032
Week8,B6	0	0.006	0.003	0.004	0.044	0.006	0.044	0.009	0.008
Week9,B6	0.016	0.026	0.033	0.006	0.077	0.01	0.084	0.015	0.016
Week10,B6	0.019	0.036	0.05	0.011	0.073	0.006	0.048	0.013	0.016
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B7	0.012	0.024	0.017	0.005	0.017	0.002	0.012	0.003	0.063
Week7.B7	0.014	0.024	0.028	0.009	0.02	0.002	0.012	0.005	0.08
Week8.B7	0.012	0.023	0.013	0.006	0.023	0.003	0.016	0.004	0.05
Week9.B7	0.013	0.028	0.024	0.006	0.024	0.004	0.017	0.004	0.062
Week10.B7	0.02	0.037	0.041	0.01	0.029	0.004	0.019	0.004	0.093

Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B10	0.0028	0.005	0.144			
Week2.B10	0.006	0	0.024			
Week3.B10	0.003	0.003	0.052			
Week4.B10	0.002	0	0.051			
Week5.B10	0.0028	0.005	0.068			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.B11	0.004	0.002				
Week2.B11	0.006	0.002				
Week3.B11	0.003	0.005				
Week4.B11	0.007	0.003				
Week5.B11	0.001	0.005				
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B1	0.001	0.004	0.081			
Week7.B1	0	0.006	0.046			
Week8.B1	0.003	0.002	0.036			
Week9.B1	0.003	0.005	0.036			
Week10.B1	0	0	0.125			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B2	0.008	0.013	0.302			
Week7.B2	0	0	0.531			
Week8.B2	0	0.003	0.37			
Week9.B2	0.002	0.005	1.155			
Week10.B2	0	0	0			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B3	0.003	0	0.115			
Week7.B3	0	0	0.055			
Week8.B3	0.001	0	0.044			
Week9.B3	0.001	0.002	0.038			
Week10.B3	0.001	0.002	0.246			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B4	0	0	0.198			
Week7.B4	0.022	0.02	0.185			
Week8.B4	0	0.003	0.095			
Week9.B4	0.003	0.002	0.171			
Week10.B4	0.002	0.003	0.582			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B5	0.002	0.005	0.076			
Week7.B5	0.002	0.006	0.101			
Week8.B5	0.002	0.003	0.051			
Week9.B5	0.001	0.003	0.097			
Week10.B5	0	0.005	0.086			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6,B6	0.008	0.005	0.07			
Week7,B6	0.008	0.023	0.145			
Week8,B6	0.009	0.005	0			
Week9,B6	0.008	0.005	0.076			
Week10,B6	0.008	0.005	0.213			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B7	0	0.007	0.044			
Week7.B7	0	0.004	0.118			
Week8.B7	0	0.005	0.076			
Week9.B7	0.002	0.008	0.105			
Week10.B7	0.002	0.006	0.68			

Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B8	0.013	0.026	0.008	0.006	0.022	0.003	0.016	0.004	0.002
Week7.B8	0.005	0.02	0.012	0.005	0.024	0.005	0.014	0.003	0.032
Week8.B8	0.012	0.011	0.006	0.006	0.022	0.003	0.02	0.005	0.008
Week9.B8	0.013	0.014	0.021	0.005	0.025	0.002	0.016	0.005	0.02
Week10.B8	0.014	0.023	0.025	0.004	0.029	0.002	0.021	0.004	0.03
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B9	0.012	0.012	0.0067	0.008	0.025	0.0036	0.018	0.003	0.0256
Week7.B9	0.005	0.024	0.012	0.006	0.025	0.0039	0.012	0.004	0.032
Week8.B9	0.004	0.023	0.003	0.007	0.022	0.004	0.018	0.004	0.008
Week9.B9	0.01	0.023	0.014	0.0076	0.022	0.005	0.012	0.005	0.01
Week10.B9	0.045	0	0.016	0.013	0.022	0.005	0.0123	0.004	0.01
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B10	0.004	0.078	0.0411	0.008	0.065	0.03	0.026	0.294	0.21
Week7.B10	0.07	0.094	0.032	0.006	0.013	0.027	0.023	0.268	0.016
Week8.B10	0.4	0.051	0.028	0.005	0.036	0.026	0.016	0.196	0.139
Week9.B10	0.051	0.073	0.035	0.005	0.178	0.036	0.022	0.244	0.17
Week10.B10	0.036	0.051	0.026	0.003	0.007	0.023	0.012	0.134	0.091
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.B11	0.014	0.04	0.031	0.0078	0.003	0.013	0.004	0.02	0.019
Week7.B11	0.023	0.043	0.058	0.001	0.0049	0.011	0.004	0.018	0.002
Week8.B11	0.017	0.056	0.004	0.007	0.003	0.021	0.0032	0.021	0.018
Week9.B11	0.0176	0.014	0.014	0.0074	0.004	0.022	0.005	0.002	0.016
Week10.B11	0.013	0	0.016	0.004	0.004	0.021	0.004	0.006	0.13
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week1.BC	0	0	0.014	0.002	0.012	0.001	0	0	0
Week2.BC	0	0	0.015	0.002	0.012	0	0	0	0
Week3.BC	0	0	0.015	0.002	0.011	0.001	0	0	0
Week4.BC	0	0	0.015	0.002	0.011	0	0.001	0	0
Week5.BC	0	0	0.015	0.002	0.012	0	0	0	0
Build	isobutane	n-butane	acetone	benzene	toluene	eth benze	xylenes	1,2 4 tmb	1,1,1 tce
Week6.BC	0	0	0.014	0.004	0.006	0	0.001	0	0
Week7.BC	0	0	0.015	0.004	0.006	0	0	0	0
Week8.BC	0	0	0.015	0.003	0.005	0.001	0.001	0	0
Week9.BC	0	0	0.015	0.004	0.006	0	0	0	0
Week10.BC	0	0	0.015	0.004	0.006	0	0	0	0

Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B8	0.002	0.0023	0.084			
Week7.B8	0	0.0021	0.054			
Week8.B8	0.002	0.001	0.03			
Week9.B8	0.002	0.0024	0.046			
Week10.B8	0.001	0.005	0.092			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B9	0.0024	0.013	0.081			
Week7.B9	0.0089	0	0.045			
Week8.B9	0.0021	0.003	0.032			
Week9.B9	0.0023	0.002	0.034			
Week10.B9	0	0.003	0.112			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B10	0.033	0.024	0.315	0.012	0.101	0.137
Week7.B10	0.029	0.01	0.286	0.01	0.086	0.121
Week8.B10	0.022	0.008	0.326	0.009	0.063	0.093
Week9.B10	0.026	0.013	0.326	0.011	0.075	0.102
Week10.B10	0.014	0.005	0.248	0.007	0.042	0.061
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.B11	0.003	0.005	0.085			
Week7.B11	0.022	0	0.046			
Week8.B11	0.002	0.005	0.043			
Week9.B11	0.001	0.0023	0.038			
Week10.B11	0.002	0.0024	0.021			
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week1.BC	0	0	0	0	0	0
Week2.BC	0	0	0	0	0	0
Week3.BC	0	0	0	0	0	0
Week4.BC	0	0	0	0	0	0
Week5.BC	0	0	0	0	0	0
Build	a-pinene	limonene	Ethanol	octane	nonane	decane
Week6.BC	0	0	0	0	0	0
Week7.BC	0	0	0	0	0	0
Week8.BC	0	0	0	0	0	0
Week9.BC	0	0	0	0	0	0
Week10.BC	0	0	0	0	0	0

APPENDIX 7.1



DATA from the QUANTITATIVE and QUALITATIVE ANALYSIS (TVOCs, TEMPERATURE, HUMIDITY and BUILDING POPULATION)

7.1 Data from the Quantitaive and Qualitative Analysis (TVOCs, Temperature, Humidity and Building Population)

Outlined in this appendix are the results of the quantitative and qualitative analysis in this Tasmanian study. The TVOC, temperature, humidity and population data listed are the weekly averages for all buildings (11 sample buildings and 1 control) during the ten sampling weeks. TVOC concentrations are shown in mg/m^3 and the temperature, humidity and population data are shown in percentages unless stated otherwise.

Legend to Appendix 7.1

TVOC mg/m^3 -	Total volatile organic compound concentrations
Av Ind Temp -	Average indoor temperature
Av Out Min T -	Average outdoor minimum temperature
Av Out Max T -	Average outdoor maximum temperature
Av Ind Humid -	Average indoor relative humidity
Av Out Humid -	Average outdoor relative humidity
Tot Pop Samp -	Total number of individuals sampled
Pop Obs Symp -	Number of individuals with symptoms attributed to the work environment
% Sick -	Percentage of individuals with symptoms attributed to the work environment
>3 symp -	Number of individuals with three or more symptoms attributed to the work environment

Building	TVOC mg/m3	Av Ind Temp	Av Out Min T	Av Out Max T	Av Ind Humid	Av Out Humid
Week1.B1	0.2617	22	10	16	62	67
Week2.B1	0.4379	21	14	25	40	63
Week3.B1	0.3085	21.5	12	19	55	49
Week4.B1	0.2914	22	11	18	54	67
Week5.B1	0.489	22	10	19	54	60
Week1.B2	0.1488	22.5	10	16	66	67
Week2.B2	0.2244	22	14	25	40	63
Week3.B2	0.1508	21.1	12	19	58	49
Week4.B2	0.1204	22	11	18	42	67
Week5.B2	0.0847	23	10	19	43	60
Week1.B3	0.1663	24.5	10	16	70	67
Week2.B3	0.1873	22.5	14	25	40	63
Week3.B3	0.1387	22	12	19	54	49
Week4.B3	0.1411	23.5	11	18	38	67
Week5.B3	0.1637	23	10	19	42	60
Week1.B4	0.2176	21.5	10	16	69	67
Week2.B4	0.2714	21	14	25	40	63
Week3.B4	0.2403	22	12	19	51	49
Week4.B4	0.1563	22	11	18	44	67
Week5.B4	0.0961	23	10	19	43	60
Week1.B5	0.2058	23	10	16	63	67
Week2.B5	0.1941	22.5	14	25	42	63
Week3.B5	0.2826	22	12	19	51	49
Week4.B5	0.1842	24	11	18	38	67
Week5.B5	0.0649	23	10	19	44	60
Week1.B6	0.2505	23	10	16	62	67
Week2.B6	0.2601	22	14	25	42	63
Week3.B6	0.2066	22	12	19	54	49
Week4.B6	0.1645	22	11	18	43	67
Week5.B6	0.2007	22	10	19	51	60
Week1.B7	0.2773	24.1	10	16	67	67
Week2.B7	0.2127	22	14	25	42	63
Week3.B7	0.1703	22.5	12	19	48	49
Week4.B7	0.1864	23	11	18	40	67
Week5.B7	0.1547	23	10	19	46	60
Week1.B8	0.392	21.3	9.3	15.9	55	75
Week2.B8	0.548	22.5	5.3	15.9	40	59
Week3.B8	0.308	22.6	6.1	13.8	43	70
Week4.B8	1.062	22	4.9	13.9	38	79
Week5.B8	0.374	21.5	4	13.3	40	85
Week1.B9	1.934	21.8	5.4	12.9	62	79
Week2.B9	0.001	22	2.2	11.2	40	73
Week3.B9	0.352	21.8	5.7	14	50	67
Week4.B9	0.19	22	8.8	18.5	38	52
Week5.B9	0.24	22	11.7	19.1	41	57
Week1.B10	0.3663	20.5	8.6	11.9	36.7	65
Week2.B10	0.1254	20.4	6.5	12.7	35.3	66
Week3.B10	0.2279	22	7	14.7	36.6	57
Week4.B10	0.245	20.9	6	14	36.8	64
Week5.B10	0.3098	20.7	8.3	17.3	37.2	59

Building	Tot Pop Samp	Pop Obs Symp	% Sick	>3 symp
Week1.B1	10	6	60	3
Week2.B1	10	6	60	3
Week3.B1	10	6	60	3
Week4.B1	10	6	60	3
Week5.B1	10	6	60	3
Week1.B2	22	20	90	16
Week2.B2	22	20	90	16
Week3.B2	22	20	90	16
Week4.B2	22	20	90	16
Week5.B2	22	20	90	16
Week1.B3	7	6	85.7	5
Week2.B3	7	6	85.7	5
Week3.B3	7	6	85.7	5
Week4.B3	7	6	85.7	5
Week5.B3	7	6	85.7	5
Week1.B4	17	13	76.4	11
Week2.B4	17	13	76.4	11
Week3.B4	17	13	76.4	11
Week4.B4	17	13	76.4	11
Week5.B4	17	13	76.4	11
Week1.B5	7	6	85.7	4
Week2.B5	7	6	85.7	4
Week3.B5	7	6	85.7	4
Week4.B5	7	6	85.7	4
Week5.B5	7	6	85.7	4
Week1.B6	6	6	100	6
Week2.B6	6	6	100	6
Week3.B6	6	6	100	6
Week4.B6	6	6	100	6
Week5.B6	6	6	100	6
Week1.B7	17	16	94.1	12
Week2.B7	17	16	94.1	12
Week3.B7	17	16	94.1	12
Week4.B7	17	16	94.1	12
Week5.B7	17	16	94.1	12
Week1.B8	40	30	75	18
Week2.B8	40	30	75	18
Week3.B8	40	30	75	18
Week4.B8	40	30	75	18
Week5.B8	40	30	75	18
Week1.B9	5	5	100	4
Week2.B9	5	5	100	4
Week3.B9	5	5	100	4
Week4.B9	5	5	100	4
Week5.B9	5	5	100	4
Week1.B10	17	15	66.6	7
Week2.B10	17	15	66.6	7
Week3.B10	17	15	66.6	7
Week4.B10	17	15	66.6	7
Week5.B10	17	15	66.6	7

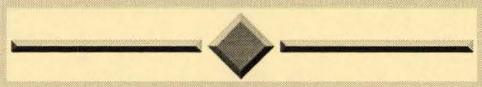
Building	TVOC mg/m3	Av Ind Temp	Av Out Min T	Av Out Max T	Av Ind Humid	Av Out Humid
Week1.B11	0.277	19.2	5.6	11.8	36.5	91
Week2.B11	0.302	21.3	4.9	14.3	30	71
Week3.B11	0.13	19.2	10.2	15	35	90
Week4.B11	0.132	19.4	5.8	19.2	36	71
Week5.B11	0.222	18.5	8.6	20	37	57
Week6.B1	0.278	25.2	5.6	11.8	20	91
Week7.B1	0.269	22	4.9	14.3	23	71
Week8.B1	0.208	21	10.2	15	20	90
Week9.B1	0.193	20	5.8	19.2	20	71
Week10.B1	0.33	23	8.6	20	36	57
Week6.B2	0.603	20	5.6	11.8	39	91
Week7.B2	0.603	19	4.9	14.3	25	71
Week8.B2	0.459	19	10.2	15	24	90
Week9.B2	1.424	19	5.8	19.2	24	71
Week10.B2	0.012	21	8.6	20	20	57
Week6.B3	0.229	22	5.6	11.8	30	91
Week7.B3	0.151	21	4.9	14.3	32	71
Week8.B3	0.1345	21	10.2	15	30	90
Week9.B3	0.151	19	5.8	19.2	32	71
Week10.B3	0.421	24	8.6	20	30	57
Week6.B4	0.349	19.2	5.6	11.8	26	91
Week7.B4	0.712	19	4.9	14.3	23	71
Week8.B4	0.269	19	10.2	15	33	90
Week9.B4	0.2986	18	5.8	19.2	33	71
Week10.B4	0.736	17	8.6	20	26	57
Week6.B5	0.228	21	5.6	11.8	22	91
Week7.B5	0.282	20	4.9	14.3	25	71
Week8.B5	0.156	19	10.2	15	30	90
Week9.B5	0.222	22	5.8	19.2	24	71
Week10.B5	0.221	24.8	8.6	20	32.3	57
Week6,B6	0.246	26	5.6	11.8	22	91
Week7,B6	0.356	19	4.9	14.3	23	71
Week8,B6	0.138	18	10.2	15	28	90
Week9,B6	0.372	21	5.8	19.2	30	71
Week10,B6	0.498	23.1	8.6	20	44	57
Week6.B7	0.206	24	5.6	11.8	18	91
Week7.B7	0.316	21.5	4.9	14.3	22	71
Week8.B7	0.231	18	10.2	15	30	90
Week9.B7	0.297	21.5	5.8	19.2	30	71
Week10.B7	0.945	24.3	8.6	20	20	57
Week6.B8	0.1883	23	5.6	11.8	23	91
Week7.B8	0.1761	20	4.9	14.3	35	71
Week8.B8	0.126	18	10.2	15	30	90
Week9.B8	0.1714	20	5.8	19.2	43	71
Week10.B8	0.25	23	8.6	20	28	57
Week6.B9	0.2103	23	5.6	11.8	76	91
Week7.B9	0.1778	21	4.9	14.3	54	71
Week8.B9	0.1301	19	10.2	15	79	90
Week9.B9	0.1469	19	5.8	19.2	54	71
Week10.B9	0.2423	22.5	8.6	20	40	57
Week6.B10	1.3781	22	5.8	10.7	50	72
Week7.B10	1.091	21	5.4	10	42	60
Week8.B10	1.418	22	4.2	13.2	56	82

Building	Tot Pop Samp	Pop Obs Symp	% Sick	>3 symp
Week1.B11	3	3	100	3
Week2.B11	3	3	100	3
Week3.B11	3	3	100	3
Week4.B11	3	3	100	3
Week5.B11	3	3	100	3
Week6.B1	4	4	100	3
Week7.B1	4	4	100	3
Week8.B1	4	4	100	3
Week9.B1	4	4	100	3
Week10.B1	4	4	100	3
Week6.B2	9	9	100	8
Week7.B2	9	9	100	8
Week8.B2	9	9	100	8
Week9.B2	9	9	100	8
Week10.B2	9	9	100	8
Week6.B3	6	6	100	5
Week7.B3	6	6	100	5
Week8.B3	6	6	100	5
Week9.B3	6	6	100	5
Week10.B3	6	6	100	5
Week6.B4	10	10	100	9
Week7.B4	10	10	100	9
Week8.B4	10	10	100	9
Week9.B4	10	10	100	9
Week10.B4	10	10	100	9
Week6.B5	7	6	85.7	5
Week7.B5	7	6	85.7	5
Week8.B5	7	6	85.7	5
Week9.B5	7	6	85.7	5
Week10.B5	7	6	85.7	5
Week6,B6	9	6	66.6	5
Week7,B6	9	6	66.6	5
Week8,B6	9	6	66.6	5
Week9,B6	9	6	66.6	5
Week10,B6	9	6	66.6	5
Week6.B7	13	11	84.6	9
Week7.B7	13	11	84.6	9
Week8.B7	13	11	84.6	9
Week9.B7	13	11	84.6	9
Week10.B7	13	11	84.6	9
Week6.B8	40	30	75.6	18
Week7.B8	40	30	75.6	18
Week8.B8	40	30	75.6	18
Week9.B8	40	30	75.6	18
Week10.B8	40	30	75.6	18
Week6.B9	5	5	100	4
Week7.B9	5	5	100	4
Week8.B9	5	5	100	4
Week9.B9	5	5	100	4
Week10.B9	5	5	100	4
Week6.B10	17	15	88.2	15
Week7.B10	17	15	88.2	15
Week8.B10	17	15	88.2	15

Building	TVOC mg/m3	Av Ind Temp	Av Out Min T	Av Out Max T	Av Ind Humid	Av Out Humid
Week9.B10	1.367	22	5.9	14.8	41	63
Week10.B10	0.76	19	10	14.8	32	61
Week6.B11	0.2448	19	6.4	16.2	18	53
Week7.B11	0.2329	19	9	17	35	58
Week8.B11	0.2002	21	6.4	16.4	40	59
Week9.B11	0.1433	20	4.7	15	35	54
Week10.B11	0.2243	23	7.8	14.2	22	62
Week 1-5 BC	0.029	22	13	18.5	50	64
Week 6-10 BC	0.025	19	6	15	32	60

Building	Tot Pop Samp	Pop Obs Symp	% Sick	>3 symp
Week9.B10	17	15	88.2	15
Week10.B10	17	15	88.2	15
Week6.B11	3	3	100	3
Week7.B11	3	3	100	3
Week8.B11	3	3	100	3
Week9.B11	3	3	100	3
Week10.B11	3	3	100	3
Week 1-5 BC	3	0	0	0
Week 6-10 BC	3	0	0	0

APPENDIX 7.2



DATA from the BUILDING QUESTIONNAIRE

7.2 Data from the Building Questionnaire

Outlined in this appendix are the results of the building information questionnaire used in the qualitative analysis for this Tasmanian study.

Legend to Appendix 7.2

Question number:

1 Year building completed?
2 What are the floors covered with?
3 What are the walls covered/treated with?
4 What does the wall consist of (interior side of insulation?
5 What does the ceiling consist of?
6 Room density
7 Overall rating for room density
8 Is there any mould growth in the building?
9 Are there any damp spots on the walls?
10 How are the rooms lit?
11 Is there a solar shading device?
12 Is there any solar gain during the course of the day?
13 How are the rooms heated?
14 How is the heating controlled?
15 Is the air temperature reduced outside working hours?
16 What is the furniture made of in a typical room?
17 The age of the furniture?
18 Are there any curtains/textiles/plastic window shading?
19 When were they last washed/dry cleaned?
20 Are there any large green plants?
21 Which rooms have extensive use of solvents etc?
22 How are the rooms ventilated?
23 Is smoking generally allowed?
24 Only allowed in separately ventilated rooms?
25 How often are the rooms cleaned?
26 What type of air conditioning system is in use?
27 Does a air system use a cooling tower etc?
28 Air exchange rate
29 Lowest outdoor air supply per person for a typical room

30 Where are the outlets placed?

31 Where are the exhaust terminals placed?

32 Is there recirculation etc?

33 How is the ventilation system operated?

34 Operating rates

35 Is there an operation and maintenance programme?

36 Who is responsible for operation and maintenance?

37 Have the following components been serviced?

Responses

Y Yes

N No

1 Option 1

2 Option 2

3 Option 3

4 Option 4

5 Option 5

Question	1	2	3	4	5	6M	6T	6L	7	8
Building 1	2	2	3	2	2	4	8	18	3	N
Building 2	2	2	2	3	5/6	7/5	9	20	3	N
Building 3	2	2	2	3	5/6	8	6	36	2	N
Building 4	1	2	2	3	5/6	11	18	23	1	Y
Building 5	2	2	2	3	5/6	9	12	15	2	N
Building 6	1	2	1/2	3/2	5/6	4	15	40	2	N
Building 7	2	2	2	3	5	5	9	20	3	N
Building 8	1	2	2	3	5	6	10	22	1	N
Building 9	1	2	2	3	5	3	6	14	3	N
Building 10	1	2	2	3	5	3	4	15	4	Y
Building 11	2	2	2	3	2	4	5	10	1	N
Control	2	2	2	3	5/6	9	12	23	3	N

Question	9	10	11.I	11.O	12	13	14	15	16.1	16.2
Building 1	N	6	Y	N	Y	5	4	N	1	
Building 2	N	6	Y	N	N	5	5			1
Building 3	N	6	Y	N	Y	6	4	Y	1	1
Building 4	Y	6	Y	N	Y	3	3	Y	1	1
Building 5	Y	6	Y	N	Y	5	4	Y	1	1
Building 6	Y	6	Y	N	Y	5	3	Y	1	1
Building 7	N	6	Y	N	Y	5	4	Y	1	1
Building 8	Y	6	Y	N	N	5	4	Y	1	1
Building 9	N	6	Y	N	Y	5	4	Y	1	1
Building 10	Y	6	Y	N	N	5	4	Y	1	1
Building 11	N	6	Y	N	N	5	4	Y	1	1
Control	N	6	Y	N	Y	5	4	Y	1	1

Question	16.3	16.4	16.5	17	18	19	20	21.1	21.2	21.3
Building 1	1	1	1	1	Y					1
Building 2	1	1		1/2	Y					1
Building 3	1	1		1	Y		1			1
Building 4	1			1	Y	96	1			1
Building 5		1		1	Y			1	1	1
Building 6	1			1	Y		1		1	1
Building 7				1	Y	97			1	1
Building 8	1			1	Y			1		1
Building 9				1	Y	97				1
Building 10				1	Y	97		1	1	1
Building 11	1			1	Y	96				1
Control	1	1		1	Y	97	1			1

Question	21.4	21.5	21.6	22	23	24	25.1	25.2	25.3	25.4
Building 1		1		3	N	N	M			D
Building 2	1	1		1	N	N	D			2
Building 3	1	1		1	N	N	D			D
Building 4	1	1		3	N	N	2			2
Building 5	1	1		3	N	N	2			2
Building 6	1	1		3	N	N	2		D	D
Building 7	1	1		3	N	N	2			D
Building 8	1	1		3	N	N	2			D
Building 9	1	1		3	N	N	2			D
Building 10	1	1		1	N	N	2			D
Building 11	1	1		1	N	N	2			D
Control	1	1		3	N	N	2			D

Question	25.5	25.6	25.7	26	27	28	29	30	31	32
Building 1			M	4	2	3	10	1	1	
Building 2				N						
Building 3				1	2			1	1	1
Building 4				1	1		10	1	1	1/3
Building 5				2	2	4	10	1	1	3
Building 6				2	1	3	10	1	1	1
Building 7				1	2	4	10	1	1	1
Building 8				1	2	4	10	1	1	1
Building 9				1	2	4	10	1	1	1
Building 10				6	2					
Building 11				2	2	4	10	1	3	1
Control				1	2	4	10	1	1	1

Question	33	34F	34R	34S	0.34	34A	35	36	37
Building 1	3	1	1	1			Y	TC	7
Building 2	2		1						
Building 3	3	1	1	1			Y	EA	7
Building 4	3	1	1	1	1	1	Y	UC	5
Building 5	3	1	1	1			Y	UC	1
Building 6	3	1	1	1			Y	AM	7
Building 7	3	1	1				Y	UC	1
Building 8	3	1	1				Y	EA	1/5
Building 9	3	1	1				Y	EA	1/5
Building 10									
Building 11	3	1	1				Y	LC	1/5
Control	3	1	1	1	1	1	Y	EA	1/5